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## EDITORIAL · REDAKSIONEEL

### PENICILLINASE

#### AN AID IN THE TREATMENT OF AN IATROGENIC HAZARD

It has been estimated that 440 tons of penicillin are manufactured annually in the U.S.A.[1] This averages out at a consumption rate of 3,000,000 units per American citizen. It is hardly surprising, therefore, to learn of the progressively increasing incidence of penicillin reactions.

The routes through which sensitivity may occur are legion. As Zimmerman has pointed out, they include

'previous oral, inhalation, suppository, insufflation, inunction, bougie, drop, spray, troche, or parenteral therapeutic penicillin. There is no means of contact, no body orifice, and no vehicle which is overlooked among the 300 odd penicillin preparations . . . Sensitivity may also be due to previous infection with superficial fungi secreting penicillin on the skin or to the constant ingestion of small quantities of penicillin in milk contaminated with penicillin, from the treatment of cows for mastitis. There is inadvertent exposure to small parenteral doses of penicillin in improperly cleansed syringes used for other injections. In polio vaccine and adenovirus vaccine manufacture, penicillin is present (200 U/ml.) as a bactericidal agent in the monkey kidney culture medium used for growing these viruses. Usually 10–35 U/ml. are present in the finished vaccine. Appreciable amounts of penicillin are inhaled by hospital employees and by workers with the drug in pharmaceutical plants.'

1. Zimmerman, Murray C. (1960): *Enzymes in Health and Disease,* Chapter 17. Springfield: Charles C. Thomas.

### PENISILLINASE

#### 'N HULPMIDDEL BY DIE BEHANDELING VAN 'N IATROGENIESE GEVAAR

Daar word bereken dat 440 ton penisillien jaarliks in die V.S.A. berei word.[1] Dit beteken dat iedere Amerikaanse burger gemiddeld 3,000,000 eenhede per jaar verbruik, en dit is derhalwe nie verbasend om te verneem dat penisillienreaksies op 'n steeds groter skaal voorkom nie.

Die roetes waarlangs gevoeligheid hom kan openbaar, is legio. Soos Zimmerman daarop wys, sluit hulle in

‚vroeëre mondelinge, inasemings-, steekpil-, bestuiwings-, invrywings-, staaf-, druppel-, spuit-, troche-, of parenterale terapeutiese penisillien. Daar is geen kontakmanier, geen liggaamsopening, en geen draer wat oor die hoof gesien is onder die ongeveer 300 penisillienpreparate nie. Gevoeligheid kan ook die gevolg wees van 'n vroeëre infeksie met oppervlakteswamme wat penisillien op die vel afskei, of van die gedurige ingestie van klein hoeveelhede penisillien saam met melk wat met penisillien besmet is nadat koeie vir uierontsteking behandel is. Daar is onopsetlike blootstelling aan klein parenterale penisilliendosisse ten gevolge van spuite wat vir ander inspuitings gebruik en nie behoorlik skoongemaak is nie. By die bereiding van polio-entstof en adenovirusentstof is penisillien teenwoordig (200 e./ml.) as 'n bakteriedodende middel in die bobbejaannierkwekingsmiddel wat vir die groei van hierdie virusse gebruik word. 10–35 e./ml. is gewoonlik in die bereide entstof aanwesig. Heelwat penisillien

1. Zimmerman, Murray C. (1960): *Enzymes in Health and Disease,* Hoofstuk 17. Springfield: Charles C. Thomas.

Penicillin reactions may range from the trivial to the fatal and in a certain percentage of cases undergoing antibiotic treatment the latter is a very real risk which needs to be guarded against. The enzyme penicillinase acts by changing penicillin into penicilloic acid, which has no antibiotic properties and is non-antigenic. It is therefore a specific method for inactivating penicillin and thus stopping the perpetuation of its allergic effects.

The availability of penicillinase does not mean that the general methods of treating the anaphylactic crisis can be abandoned. All the usual emergency measures must be resorted to, including the use of antihistaminic preparations, steroids, etc. The penicillinase effect is observed after hours, whereas the allergic emergency may develop in a matter of seconds. Davidson[2] has outlined a routine method of using an antihistamine in combination with penicillin to prevent allergic reactions. Zimmerman has pointed out that

' the palliative use of steroids, ACTH, epinephrine, and antihistamines to relieve the symptoms of less acute allergic reactions while penicillinase is destroying the causative antigen is just as proper as the use of anodynes to relieve the discomfort of any other disease process. There may even be a synergistic effect, the steroids relieving edema and inflammation, allowing penicillinase to penetrate sooner into affected tissues.'

Valuable as penicillinase is in the field of treating allergic reactions to penicillin, it must be appreciated that clear-cut failures which are unequivocal and at present inexplicable, have been observed in under 20% of cases.

About one-third of the patients complained of slight local reactions at the site of injection and some 10% of patients have reacted with mild, self-limited febrile responses which disappeared within a few hours. The enzyme in itself is capable of producing an anaphylactic reaction. Despite these facts, it remains the most specific agent available for the treatment of penicillin reactions, and affords greater therapeutic freedom ' to the physician who is confident of his ability to cope with penicillin reactions by the use of penicillinase.'

2. Davidson, A. (1957): Med. Proc., **3**, 168.

word ingeasem deur hospitaalwerknemers, en deur persone wat met die middel in farmaseutiese inrigtings werk.'

Penisillienreaksies wissel van die onbenullige tot die noodlottige, en by 'n sekere persentasie pasiënte wat antibioties behandel word, is laasgenoemde 'n baie wesenlike gevaar waarteen gewaak moet word. Die ensiem penisillinase werk deur penisillien in penisilloïensuur te verander. Laasgenoemde het geen antibiotiese eienskappe nie en is nie-antigenies. Dit is derhalwe 'n spesifieke metode om penisillien te inaktiveer en dus die voortsetting van sy allergiese effekte stop te sit.

Die beskikbaarheid van penisillinase beteken nie dat daar afgesien kan word van die algemene metodes vir die behandeling van die anafilaktiese krisis nie. Al die gewone noodmaatreëls moet toegepas word, insluitende die gebruik van antihistamien, steroïede, ens. Die effek van penisillinase word eers na etlike ure waarneembaar, terwyl die allergiese noodtoestand binne enkele sekondes kan ontstaan. Davidson[2] het 'n beskrywing verstrek van 'n roetine-metode om 'n antihistamien saam met penisillien te gebruik om allergiese reaksies te voorkom. Zimmerman wys daarop dat

‚die versagtende gebruik van steroïede, ACTH, epinefrien en antihistamiene vir die verligting van die simptome van minder akute allergiese reaksies onderwyl penisillien besig is om die oorsaaklike antigeen te vernietig, net so raadsaam is soos die gebruik van pynstillende middels om die ongerief van enige ander siekteproses te verlig. Daar kan selfs 'n sinergistiese effek wees, gesien die feit dat die steroïede edeem en ontsteking verlig, waardeur die penisillinase dan toegelaat word om vinniger tot die aangetaste weefsels deur te dring.'

Hoe waardevol penisillinase ook al is vir die behandeling van allergiese reaksies op penisillien, moet daar besef word dat duidelik mislukkings wat ondubbelsinnig en op die oomblik onverklaar is, in minder as 20% van die gevalle waargeneem word.

Ongeveer een-derde van die pasiënte het gekla oor geringe plaaslike reaksies by die inspuitingsplek, en ongeveer 10% van die pasiënte het gereageer met ligte, selfbeperkende koorsreaksies wat binne 'n paar uur verdwyn het. Die ensiem self is in staat om 'n anafilaktiese reaksie te veroorsaak. Ten spyte hiervan bly dit die mees spesifieke tans beskikbare middel vir die behandeling van penisillienreaksies, en dit bied groter terapeutiese vryheid ‚aan die geneesheer wat vertroue het in sy vermoë om penisillienreaksies met behulp van penisillinase teë te werk.'

2. Davidson, A. (1957): Med. Byd., **3**, 168.

## ABSTRACTS

### THE EFFECT OF HYPERTENSION II IN NORMOTENSIVES

The pressor effect of hypertension II was tested in fairly young patients with no circulatory disease and normal blood pressure, in order to ascertain whether the substance was suitable for clinical use. The dose : effect ratios were determined by direct measurement of the blood pressure, the ECG being recorded at the same time. A cardiac catheter was also used in some cases.

The observations confirmed that hypertension II has an action similar to that of noradrenaline, but increases the blood pressure more markedly. The effect is due to an increase in peripheral resistance, while the cardiac minute volume remains virtually

unchanged. The vasoconstriction affects not only cutaneous and muscular vessels, as well as the splanchnic bed, but also extends to the pulmonary vessels. The increase in pressure is dependent on the dosage, the rise in diastolic being considerably less marked than the rise in systolic. The hypertension was found to be accompanied by increasing bradycardia which was occasionally associated with temporary arrhythmia. By means of continuous drip infusion the increase in blood pressure can be easily maintained for several hours without tachyphylaxis developing. After the infusion has been stopped, the blood pressure soon returns to normal.

The investigations showed that synthetic hypertension II is a physiological type of substance which exerts a marked pressor effect. It is well tolerated and can be used with success in states of collapse and shock.

[Lichtlen, P., Bühlmann, A. and Schaub, F. (1959): Cardiologia (Switz.), **35**, 139].

### UTERINE RETROFLEXION AS AN ANTHROPOLOGICAL CHARACTERISTIC

Various questions connected with the significance of uterine retroflexion in female pathology have at one time or another led to heated discussions. Accordingly, the author investigated the incidence of uterine retroflexion among 463 Indonesian women in West Java. The women were quite well-to-do, hardly any were called upon to perform heavy physical labour, and most were adequately nourished.

Retroflexion was found to be present in 88% of the non-pregnant women, and in 54% of women in early pregnancy. In the author's view, retroflexion in Indonesian women is not due to exogenous factors, such as malnutrition, exhausting diseases, severe physical exertion, or traumata, but rather to endogenous influences.

It is interesting to note that, in about one third of the Indonesian women examined, retroflexion changed to anteflexion during early pregnancy. Indeed, this change in position is actually a valuable aid in diagnosing pregnancy in Indonesian women.

The author is unable to offer any explanation for the change from retroflexion to anteflexion.

[Ravesteyn, T. L. W. van (1959): Trop. Geograph. Med. (Nethl.), **11**, 61].

### THE MENOCYTES

Morphologically, the menocytes are closely connected with the histiocytes and are doubtless derived from them. They contain heparin, or its precursor, and are found in very large numbers in the endometrium during menstruation and in the first few months of pregnancy. It may be assumed that the menocytes are *partly* responsible for the fact that menstrual blood, as well as maternal blood when the placenta is being formed, does not coagulate.

Very few, if any, menocytes are to be found in the follicular phase. Following ovulation they increase in number. Before or during menstruation large numbers can be observed in the endometrium. The increase in the number of menocytes depends, as a rule, on progesterone. If an artificial cycle is provoked with oestrogens alone, the uterine mucosa is poor in menocytes when withdrawal bleeding sets in (yet the incoagulability of the blood is not altered). Menocytes occur in greater numbers following combined therapy with oestrogen and progesterone. They do not, however, seem to be absolutely dependent on progesterone because they are sometimes also observed in cases of metrorrhagia or in the absence of a corpus luteum. Nevertheless, menocytes never occur so plentifully as in normal menstruation and in the decidua during the first few months of pregnancy. Menocytes are also found in patients with functional endometriosis. They are absent, however, in cases of uterine carcinoma, moles, polypi and endometritis.

[Yaneva, H. and Netter, A. (1959): Rev. Franç. Études Clin. Biol., **4**, 553].

### ADRENOCORTICAL INSUFFICIENCY IN AN INFANT WHOSE MOTHER HAD RECEIVED PREDNISONE TREATMENT

The author reports on a case of passive, temporary adrenocortical insufficiency in a 9-day-old infant. The child had been admitted to the Children's Clinic with the diagnosis of 'loss of appetite and dyspepsia.' The past history revealed that towards the end of her pregnancy the mother had received 40 mg. Ultracorten daily for a matter of weeks because of rheumatoid arthritis and rheumatic carditis. Following treatment with human milk and Diarex, the child's condition improved; a transfusion helped it through the critical phase. When it was 23 days old, however, a very severe clinical picture developed (collapse, violent diarrhoea and dehydration) with the result that the child had to receive transfusions and continuous drip infusions.

There was no evidence of an infectious component or an allergic cause, nor could any malformation or alimentary noxious agent be found. A sudden bout of violent vomiting led the author to suspect 'pseudo-pyloric stenosis.' The urinary excretion of sodium chloride was therefore checked. It was found to be high, thus suggesting a diagnosis of adrenocortical insufficiency. This diagnosis was confirmed with the aid of the ACTH test and other reactions. The success of cortisone treatment (initially 5 mg. daily, then 10 mg.) and the results of subsequent ACTH tests performed for control purposes, provided further conclusive evidence of adrenocortical insufficiency. After one month, the cortisone could be slowly discontinued and the child, having recovered, was discharged.

[Hottinger, A. (1959): Schweiz. Med. Wschr., **89**, 419].

### PULMONARY INFARCTION IN CARDIAC CASES

Pulmonary infarction is present in about 25% of cardiac patients, as evaluation of a large number of autopsy findings has shown. Patients with myocardial infarction, in particular, are prone to this complication. Cardiac patients frequently do not tolerate digitalis so well after the onset of pulmonary infarction.

[Franco Browder, S., Pérez Olea, J. and de Francisco, A. (1959): Arch. Inst. Cardiol., México, **29**, 31].

# A SURVEY OF ANAESTHESIA IN GREAT BRITAIN

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(*Concluded from p.* 524)

*The I.C.I. Laboratories at Alderley Edge, near Manchester.* A visit to the laboratories is highly recommended. All the experimental work on Halothane is done here under the guidance of Raventos. Groups of anaesthetists are invited from time to time to visit these laboratories and are given an extremely interesting and enjoyable outing.*

A whole day is taken up with a tour of the laboratories and a detailed explanation of the experimental work performed. Apart from the experiments carried out with Halothane, which is always tried out on mice before larger animals are used, I found the biological aspect rather illuminating.

Mice must be bacteria-free before they can be used for certain experimental purposes. It took these workers about 2 years before they could attain this condition. It means, in the first place, that all the workers have to be completely disinfected before entering the laboratories. They have to change into sterile garments and are subjected to all kinds of disinfecting rays. The laboratories are out of bounds and the precautions taken remind one of an atomic plant.

The mice, at term, are delivered by caesarean section and the newborn are artificially fed. This is done by passing a stomach tube every 3 hours.

*Manchester.* This town was quite a revelation in anaesthesia. Here Michael Johnstone, who was the first person to use Fluothane on a big scale, gave me a real surprise.

The first case he anaesthetized was for a nephrectomy. It was noticed that the anaesthetic trolley did not contain any syringes and without any ado the mask was placed on the face of the patient who was told that he was going to smell some sweet smelling gas; 5 litres of $O_2$ with 5% $CO_2$ were given for 20 seconds followed by 4% Fluothane and $O_2$ for another 20 seconds. The Fluothane was then stepped up to 6% for 20 seconds, then to 8% for 20 seconds and finally to 10% for 30 seconds. At the end of this time the patient was completely apnoeic and relaxed. The patient was then intubated. $O_2$ was given until the patient started breathing again and then the Waters cannister was put into circuit and Fluothane again added in concentration between 4–10% for the rest of the operation. Until the patient was intubated, the bag was not touched at all, but afterwards respiration was assisted from time to time. The patient was excellently relaxed and very little bleeding took place. The pulse rate was 120 per minute and there was only a slight fall in blood pressure. There was rapid emergence from the anaesthetic in this case. The premedication used by Michael Johnstone consists of 50 mg. Pethidine, 50 mg. of an antihistamine and no atropine. He feels that atropine increases the pulse rate and the bleeding and has no place in his cases.

The next case was one of carcinoma of the bladder in a cachectic patient 70 years old. A new bladder was fashioned out of a loop of ileum and the ureters were transplanted. The operation lasted 5 hours and the same technique was used as in the first case. Here the blood pressure dropped from 140 mm. to 75 mm. Hg and the pulse rate slowed to 40 per minute. The ECG tracings were normal and the anaesthetic technique was not changed in any way on account of the bradycardia. ECG monitoring was carried out in every case. This case also was well relaxed and remained a good colour. The arousal period was, however, prolonged.

Another case was an anaesthetic for a prostatectomy in a patient 76 years old. Johnstone feels that the analgesic effect of Fluothane wears off so rapidly that these patients are in great pain before relief can be given in the wards. In addition to the usual technique a new powerful analgesic called Sernyl was used.

Although this new drug has a prolonged analgesic effect, it has distinct disadvantages. It can cause mental changes such as amnesia and hallucinations for a day or two. This patient was well relaxed and no bleeding whatsoever occurred, so much so that the surgeon became very worried and wanted to know if the patient had a blood pressure.

Johnstone has been using Fluothane + oxygen for the last 2 years and prefers it to the usual balanced anaesthetic. He maintains that by using only Fluothane and oxygen he is in a better position to judge the effect of his anaesthetic than are others who use Pentothal, relaxants, nitrous oxide and other analgesics. The analgesic effect of nitrous oxide is often due to anoxia and the Pentothal may be the cause of a severe drop in blood pressure. A combination of these drugs may also have other unexplained effects on the patient. He feels happier with a one-drug anaesthesia. In his hands he certainly produces ideal operating conditions for the surgeon with this technique. The Fluotec he uses was built especially for him and is the only one of its kind.

Johnstone is not in the habit of taking blood pressure readings and prefers to be guided by the ECG. Extrasystoles did occur in this case,

* A visit by appointment can be arranged by indicating to the ICI organization (or its representatives in South Africa) the practitioner's wish to visit the laboratories.—*Editor.*

but as they were of the auricular variety they were ignored.

*Liverpool,* a neighbour of Manchester, feels quite differently about Fluothane. Here the standard anaesthetic consists of Pentothal, a relaxant and nitrous oxide. As enthusiastic as Michael Johnstone is about Fluothane, so is Cecil Gray about nitrous oxide.

In this very fine centre of anaesthesia it is asserted that nitrous oxide has definite advantages over any other analgesic, in that it is entirely non-toxic. The patient awakens immediately after the anaesthetic and retains a certain degree of analgesia for about 3 hours afterwards. Gray and Geddes have found by experimenting with medical students that 50% nitrous oxide with hyperventilation is an efficient analgesic. This is certainly not the case in Johannesburg, where the partial pressure of nitrous oxide in the alveoli is about 100 mm. Hg. less than at sea level. Here the concentration of nitrous oxide should be at least 70% to have the same effect. The blood concentration should reach 20% by volume of nitrous oxide.

Most of my time here was spent with Jackson Rees at the Alderhey Children's Hospital. The standard of paediatric anaesthesia as practised here was very instructive. Every case, no matter what age, is induced by the intravenous route by means of a 26 needle which is only used once and then discarded. Pentothal, in the dosage of 25 mg. per stone, is injected. A vein on the anterior surface of the wrist is used. If the baby is under one month old, Scoline is the relaxant chosen, both for the intubation and maintenance. If older, Tubarine is the only relaxant used in the dosage of 4 mg. per stone. Every case is intubated and the ventilation controlled using high flows of gases and hyperventilating with a 50/50 mixture of nitrous oxide and oxygen by the T-piece technique. This is done up to the age of 4 years and after that a to-and-fro absorber is used. After intubation, the mouth is packed with ribbon gauze, which helps to keep the tube in place and prevent it from riding on the cords and causing bruising. It is claimed that nearly 40,000 children have been intubated here without the occurrence of a single case of oedema of the glottis. Movement of the limbs during the anaesthetic is not a sign of awareness.

It must be emphasized again that the assistance given by the anaesthetic technicians here is of a very high standard. The assistant always chooses the size of tube to be used for the different babies and it always seems to be the correct one. He also knows just how to hold the arm to facilitate the very delicate intravenous injection in the neonate.

*Edinburgh.* Here I spent an interesting session with James Robertson. He was investigating the possibilities of Pressuren. He had used this drug in over 600 cases for induction. He has explored all the possible different methods of injection, such as giving it in greater concentrations rapidly, adding Procaine or cortisone to the solution, mixing it with saline and heating the solution. With none of these methods could he prevent thrombophlebitis. This complication appeared in 8% of cases. A very marked hypotension can occur when using this drug in combination with Fluothane, as illustrated by the following case:

An old man of 76 years of age was operated on for a prostatectomy; 400 mg. of Pressuren in 5% solution were injected rapidly. He was intubated after injection of 60 mg. Flaxedil and the respirations were controlled, using the closed circuit. Nitrous oxide and oxygen inhalation with 0.75% Fluothane was the maintenance anaesthetic. The blood pressure dropped to 40 mm. Hg. immediately before the enucleation of the prostate and remained at this pressure until the end of the operation, in spite of the fact that the Fluothane was stopped soon after the fall of blood pressure was discovered. The anaesthetist did not seem to be in the slightest perturbed by this extreme hypotension. At the end of the operation 4 mg. Methoxamine were given intravenously and the patient made a good recovery.

James Robertson intends abandoning the use of this steroid on account of the high incidence of thrombophlebitis.

*Newcastle-on-Tyne.* Professor Pask is the Director of Anaesthesia here. He does a great deal of research and investigation in an effort to devise new anaesthetic aids. He demonstrated his electrical pulsometer and blood pressure recorder and also the cardiac tachometer. This instrument is similar to an ECG. Instead of recording cardiac complexes, it records the pulsations on a dial. Two needles are inserted under the surface of the skin in the chest wall, one in the fifth intercostal space on the left side and the other in the third intercostal space on the right side.

All kinds of respirators are in evidence here. Some of the anaesthetic machines are fitted with miniature respirators weighing only a few pounds. Pask is the inventor of most of these so-called 'third hands.' Very little bag squeezing is done by hand at Newcastle. The feeling is that it can be done more efficiently by machine. These respirators work from the $O_2$ pressure from the mains at a pressure of 60 lb. per sq. inch. A Venturi system sucks in the gases.

*Southend-on-Sea.* A. J. Lee is a supreme performer, meticulous in everything he does, using all kinds of aids and obviously enjoying his work. Moreover, he is a great enthusiast who believes it necessary to be *au fait* with every type of useful anaesthetic technique.

To give an instance of his versatility, he demonstrated a case of abdominal hysterectomy done by the extradural method of anaesthesia.

Because of the importance of having an open vein accessible in these procedures, he always first inserts a Mitchell needle into an arm vein. He then places the patient in the lateral position and injects a little Procaine in the most convenient space in the lumbar area. With a short stout needle the skin and tissues are punctured for about ½ inch. A lumbar puncture needle marked in centimetres is inserted up to the 3 cm. mark and then tested for loss of air resistance. This was done carefully every few mm. and when the 4 cm. mark was reached there was definite loss of resistance. This is confirmed by loss of fluid resistance by injecting 2 c.c. of normal saline; 30 c.c. of 1.5% Zylocaine was then injected through the spinal needle and the patient put on to her back.

After 10 minutes the blood pressure had dropped from 120 to 100 mm. Hg.

Two tests were then done to assess the efficacy of the anaesthetic in this particular case, i.e. loss of knee jerks and, after the patient had been asked to cough, the extent of bulging of the lower abdomen. Before starting surgery 400 mg. of Evipan was given through the Mitchell needle. The patient was edentulous and would not tolerate a pharyngeal airway so the tongue was pulled forward and a roller bandage inserted into the mouth. This immediately improved the breathing. Relaxation was excellent and very little bleeding occurred.

The next case was for a cholecystectomy, and here the technique used was the usual Thiopentone, Flaxedil, nitrous oxide, $O_2$ sequence with controlled ventilation by the Blease machine. Lee prefers this to hand squeezing of the bag. He demonstrated, with the help of the Blease machine, that with hand squeezing, the pressure in the bag never drops to zero with expiration. In other words, the pulmonary pressure is always positive with this method. There was sweating of the forehead and face during this procedure, but Lee did not think that it was due to too light anaesthesia. Sweating used to occur with deep ether anaesthesia and may also be due to heat or $CO_2$ accumulation.

A tachometer was also demonstrated on this case for observation of the pulse rate.

An operation for the removal of a tumour from the chin then followed. Lee thought it would be a good idea to demonstrate his method of blind nasal intubation. He considers it the duty of every anaesthetist to keep up the practice of blind nasal intubation because of the occasions when this is the only method which can be used. He injected 200 mg. Thiopentone and then 25 mg. Scoline, sprayed the nose with 5% cocaine and then inserted a well curved nasal tube into the nostril, at the same time manipulating the larynx with the index and middle fingers of the other hand, whilst the tube engaged the glottis.

A thyroidectomy was then performed under the lytic cocktail technique after infiltration of the neck with adrenaline and saline. Intubation under Scoline, nitrous oxide and oxygen inhalation completed the anaesthetic. The result was a dry field.

The next case was a Fothergill operation. Pressuren in a 5% solution in saline, heated to body temperature, was rapidly injected through a Mitchell needle. Intubation, after Scoline and controlled ventilation with nitrous oxide, oxygen and 1.5% Fluothane inhalation was carried out. The blood pressure dropped from 150 to 100 mm. Hg., producing a bloodless field.

The technique of intermittent extradural injections was demonstrated in a case of prostatectomy. Instead of using a spinal needle a Tuohy needle was used for entering the extradural space. Once this was done a narrow Nylon tube was threaded through the Tuohy needle and the latter withdrawn. The tubing was then strapped to the patient's back and 1.5% Zylocaine injected as required.

A case of stripping varicose veins was performed under controlled ventilation by means of a non-return Ruben valve and a mask. Thiopentone was first injected through a Heller's connexion attached to a Record needle and intermittent injections of Scoline were given as required. To prevent the Rubens valve from sticking, Lee attached a Magill expiratory valve, slightly opened above the bag, to prevent too high a build-up of pressure.

To complete the list, a caudal block was performed for a sigmoidoscopy procedure.

From the description of these cases it can be gleaned that no stereotype technique is used by Lee, who keeps himself proficient in a wide variety of methods.

On the other hand, Rex Marrett at *Coventry* prefers to perfect one technique and to use it for most cases. He feels that Fluothane is such a perfect anaesthetic that, if properly used, it fulfils most of the requirements of modern surgery. He uses the closed method with the Marrett machine. The vaporizer is calibrated for the different percentage concentrations of Fluothane and by the twist of a knob either the to-and-fro or circle absorber can be used.

The following cases may serve to illustrate his technique.

A prostatectomy was performed on a 65-year-old patient. He was induced with 6 litres of nitrous oxide, 0.5 litre of $O_2$ and 0.5% Fluothane for 5 breaths and then given 2% Fluothane with 500 c.c. $O_2$. After a few minutes a pharyngeal airway was inserted, the mask tightly applied and the to-and-fro absorber put into circuit. From then onwards a close watch was kept on the bag and on the blood pressure.

The extent of movement of the bag is a good indication of the depth of anaesthesia. Breathing was spontaneous and was never assisted. The concentration of Fluothane varied between 1.5 and 3%. The blood pressure, which was taken with an oscillometer, fell from 220/126 to 150/90 mm. Hg. Ten minutes before the end of the operation the Fluothane was turned off and only nitrous oxide and oxygen in the proportion of 6:2 litres given with the absorber out of circuit. The patient remained a good colour throughout the operation and very little bleeding took place. Marrett thinks that by giving gas and oxygen for the last part of the operation, shivering, which sometimes occurs at the end of an operation under Fluothane anaesthesia, can be prevented.

The next case was a gastrectomy for carcinoma of the stomach in a 52-year-old woman. The induction was carried out with 150 mg. Thiopentone and then a flow of 500 c.c. $O_2$ per minute with 2.5% Fluothane was added to the breathing system. After a few minutes the to-and-fro absorber was put into circuit. A pharyngeal airway was inserted and a closely fitting mask applied. It was noticed that the recti were still tight when the surgeon tried to open the peritoneum. Marrett did not increase the concentration of Fluothane but admitted defeat as far as the relaxation produced by Fluothane was concerned. To overcome this, the Fluothane was shut off, 50 mg. Scoline injected and 4 litres of nitrous oxide with 2 litres of $O_2$ turned on. As soon as apnoea occurred the lungs were inflated every 15 seconds with this gas and oxygen mixture until breathing re-started. Then Fluothane 2.5% and 500 c.c. $O_2$ was again administered by the closed method. The patient remained relaxed for the rest of the operation.

Marrett maintains that once the spasm of the recti is broken, Fluothane will keep the patient relaxed. In his opinion, it is never necessary to repeat the relaxant.

It will be noticed that in none of these cases was intubation used. Marrett only intubates in head and neck, intestinal obstruction and thoracic cases. For minor operations his technique consists of 0.5% Fluothane with 6 litres nitrous oxide and 0.5 litre $O_2$ inhalation until the eyelash reflex disappears. He then continues with only gas and oxygen. The patient awakes immediately after the cessation of the anaesthetic and is ready for discharge. Marrett's simple technique of Fluothane anaesthesia impressed me as safe, efficient and economical.

*East Grinstead.* Here hypotensive anaesthetics are used for most of the plastic surgery of the head and neck. Hale Enderby demonstrated his technique for a case on which submucous resection was performed and a nasal manipulation carried out.

The patient was a fit young man of 30 years. He was given Thiopentone and 5 mg. of Decamethonium and intubated. Then 14 mg. of Ansolysen were injected. After a few minutes his blood pressure dropped from 125 mm. to 100 mm. Hg. The patient's ventilation was controlled with a fairly closed expiratory valve, with the circle absorber in circuit. An oscillometer was used for checking the systolic blood pressure every few minutes. At the commencement of the operation the patient was tilted into a deep anti-Trendelenburg position and the blood pressure dropped to 60 mm. Hg. When the deflections increased in amplitude it meant that the blood pressure was increasing. Pressure on the bag was increased and the blood pressure again dropped to 60 mm. Hg. After a while, the patient started straining on the tube and 75 mg. Brevedil E were given. After the operation had been in progress for 45 minutes, the blood pressure again showed signs of increasing and 1% Fluothane was then added to the anaesthetic with the result that the blood pressure again settled at 60 mm. Hg. Enderby explained this by postulating that adrenaline secretion increases with the duration of the operation and after a while increases the blood pressure by its vasoconstriction in spite of the vasodilating action of the Ansolysen. He considers that at this stage Fluothane would be the best vasodilator of all.

Needless to say, there was no bleeding at all. After the plugs and bandages had been applied, the Fluothane was stopped and pressure on the bag released. The table was straightened out and the blood pressure rose to 140 mm. Hg.

The patient, however, took quite a long time to come round in the post-operative recovery room. He was nursed by an experienced assistant, who only removed the intra-tracheal tube after half an hour. The blood pressure was taken every few minutes with an oscillometer. Because Ansolysen has rather a prolonged action, the blood pressure must be watched for a few hours after the operation.

Nearly all the other anaesthetists at this hospital use Arfonad instead of Ansolysen. Enderby, however, thinks that the latter drug maintains a smoother, prolonged, drier field than does Arfonad, which has a more irregular effect.

## SUMMARY

Although anaesthetic techniques varied in the different centres, the standard of anaesthesia is very high. Not only is the standard of technical skill impressive, but also the enthusiasm displayed by the leading exponents in this field, an enthusiasm with which they have infected those working with them.

There is no doubt that definite progress is being made in the development of modern anaesthesia. As has been pointed out, active research is being carried out throughout the various centres in Britain.

Anaesthesia has developed to such an extent since the war that there is now a tendency for anaesthetists to specialize in distinct branches. For instance, some workers confine

themselves to paediatrics, others to neurosurgery, others again to thoracic work, to hypotensive practice and even to dental anaesthesia.

More and more anaesthetists are using artificial ventilators for controlled ventilation with the advantage that the anaesthetist is free to perform other duties besides the ventilation of the patient. The tidal and minute volumes are more constant and in a prolonged anaesthetic the interference with the venous return can be minimized by using the negative phase during expiration.

Mechanical aids such as oscillometers, combined electrical pulsometers and blood pressure recording machines, tachometers and other types of electro-physical equipment are being developed.

The progress of anaesthesia is definitely on the march and further developments during the next few years can confidently be expected.

I wish to thank Dr. J. C. Nicholson, Head of the Department of Anaesthesia, Johannesburg Hospital, for his interest and helpful advice in the preparation of this paper.

# MILLI-OSMOLS MADE EASY

## SOME FUNDAMENTAL BIOCHEMICAL AND CLINICAL CONSIDERATIONS

### WITH PARTICULAR REFERENCE TO PAEDIATRICS

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(*Concluded from p.* 538)

### SOME THERAPEUTIC CONSIDERATIONS

#### ELECTROLYTE MANIPULATIONS

A knowledge of the aforementioned mechanisms of fluid loss enables one to have an intelligent appreciation of the problem of fluid and electrolyte replacement.

Total water lack (or excess) can be gauged from the knowledge that some 65% (70–80% in very young infants) of the body weight is water, i.e. 0.65 litre per Kg. of weight=total body water. This gives a reasonably accurate value if body weight is known or can be guessed fairly accurately. Deficits of water (if such exist) can then be replaced. Deficits or excesses of electrolyte are not so easily dealt with because their distribution is not uniform within the intracellular and extracellular compartments. Indeed, they differ considerably. In clinical practice one tests only the electrolytes of the extracellular fluid (via the plasma) and one can thus treat and manipulate alterations in this compartment, but this does not enable one to estimate at all accurately any changes within the intracellular space. For example, it is not unknown for a severe intracellular potassium deficiency to be present while the plasma potassium is reasonably normal. The latter does not necessarily reflect the state of the intracellular ion. It has been written authoritatively that:

'Hypotonicity affects both phases of body fluids and the amount of salt needed must be calculated on the basis of total body water.'[14]

The statement is true; the conclusion is not, for the salt is not uniformly distributed. In replenishing electrolyte losses, it is wiser, as far as Na and Cl are concerned, to consider only the extracellular compartment (20% of body weight in adults and about 40% in infants—Fig. 1). The intracellular Na is hardly more than 10 mM. per litre. The opposite is the case with K. The vast proportion of K is within the cell, so that one pays little attention to plasma K values in estimating total K losses; indeed this cannot be calculated with any degree of accuracy. Plasma K estimations merely enable one to make a reasonably intelligent guess about the intracellular status of potassium. The electrocardiogram helps to guess better.

#### FLUID REPLACEMENT

Minimal fluid requirements are easily understood if we make recourse to the earlier discussion on fluid losses.

Insensible losses are at least 40 c.c. per D.U. However during the process of metabolism, water of oxidation is invariably formed and made available to the body, the water thus formed being in the order of 10 c.c. per 100 calories metabolized; so that, in fact, basic water needs are about 30 c.c. per D.U. Nevertheless, more water is needed to go with endogenous and obligatory urea formation: some 5 mOsm urea per D.U. This will require 1.0 c.c. water per mOsm for excretion (and nearer

to 2 c.c. water per mOsm of urea in very young infants). Also needed is water for any electrolyte or protein supplied: again 1 c.c. (in adults and older babies) per mOsm of solute. This latter varies from about 12 mOsm per D.U. in fully breast-fed babies to 40 mOsm per D.U. for adult diets. The average intravenous (protein-free) electrolyte load is in the order of 25 mOsm per D.U.

Basic minimum water needs are then as follows (Table 3):

TABLE 3

| | |
|---|---|
| Insensible losses | 40 c.c. per D.U. |
| Credit: Water of oxidation | 10 c.c. per D.U. |
| | 30 c.c. per D.U. |
| Needed for 5 mOsm urea per D.U. | 5 c.c. |
| Water for (say) intravenous load of 25 mOsm per D.U. | 25 c.c. |
| Total | 60 c.c. water per D.U. |

This represents the absolute minimum water needs to excrete all solute via bladder, bowel and skin and to replenish all water losses via these agencies as well as via respiration, and this water intake will be only just sufficient to prevent hyperosmolarity of the body fluids. It provides no reserve whatever in the event of sweating, hyperpnoea, excessive bowel or bladder losses. In the absence of these factors, a subject on an intravenous drip thus requires a basic minimum of 60 c.c. water per D.U. and on this fluid intake the urine will be maximally concentrated (1 c.c. per mOsm of solute). Premature, newborn and young infants who need more than 1c.c. of water to excrete 1 mOsm of solute will need rather more than 60 c.c. per D.U. as a minimum water requirement. They will require some 50–60 c.c. per D.U. to excrete the urea plus electrolytes, so that basic minimum requirements in early infancy are nearer to 80–90 c.c. per D.U. and in adults may even be somewhat less than 60 c.c. per D.U., and such quantities of fluid will be sufficient to excrete a maximally concentrated urine but without any reserve.

## SURGERY: WATER INTOXICATION

A knowledge of such minimum water requirements is of particular value in the post-operative period when pharmacological agents are active in producing antidiuresis. Surgery itself produces an antidiuresis with the formation of a small quantity of concentrated urine. In older children and adults, ether, cyclopropane and morphine act directly on the kidney to induce antidiuresis, and they also stimulate the production of antidiuretic hormone, as does Pethidine. Whenever any of these agents have been used, there is a retention of body water with maximal concentration of solutes in the urine—to the extent of 1 c.c. water per mOsm excreted. The body thus tends to become waterlogged and until such time as the kidneys can manage to excrete as much as 4 c.c. water per mOsm of solute (the normal maximum when antidiuretic hormone is not active is 10 c.c. per mOsm), there is a serious danger of water accumulation and intoxication. Hence, postoperatively it is wise to limit intravenous water to about 60 c.c. per D.U.

Postoperative water intoxication is especially common in children[15] and, as most of the excess water enters the intracellular compartment, clinical oedema is rare. Because the blood volume is only minimally expanded, the packed cell volume is normal and the haematocrit is thus of no help in the diagnosis. Also normal are the plasma K (the normal value is too small to be appreciably reduced by plasma dilution) and urea, as well as the pulse, blood pressure and the ECG. The clue to the diagnosis is found in the considerably increased weight of the patient—bearing in mind that all sick individuals on intravenous drips lose weight from tissue breakdown: in adults, about 1 lb. weight loss daily. Examination of the extracellular fluid (via the plasma) indicates that the Na content is consistently below 120 mOsm per litre, and this reflects the even greater dilution of intracellular electrolyte by water. The low Na content must not be misinterpreted as an absolute deficiency of sodium. It is an absolute excess of water. Utilizing the formula Na X 2 = total (4 electrolytes only) osmolar concentration, then the plasma Na of 120 mEq. per litre indicates a total osmolarity of 240 mOsm per litre, and looking at this 'upside down,' there are $\frac{1{,}000}{240} = 4.2$ c.c. water per mOsm, a value in excess of the normal 3.5–3.7 c.c. water per mOsm of solute.

The symptomatology of water intoxication is primarily neuro-psychiatric. In those old enough to speak their symptoms, the first is headache. Other symptoms include vomiting, apathy or mania, confusion, inco-ordination and, later, fits and coma. In this state and shortly before it there may be extensor plantar responses and hyperactive knee jerks.[16] Aside from serial weighing and plasma biochemistry studies, the principal requirement for a diagnosis is an awareness that this condition exists and that water intoxication may result from manifold and devious means—in particular, bowel and stomach wash-outs in infancy.

Treatment is by administration of intravenous hypertonic salt: 3% saline has 1,000 mOsm of electrolyte per litre of water.

## CLYSES

Subcutaneous clyses should only be performed with circumspection. They have their hazards: osmotic ones. Dextrose 5% in water is roughly iso-osmotic with plasma, having some 300 mOsm of solute per litre of water. (This statement is easily verified by calculations based on the molecular weight of dextrose, $C_6H_{12}O_6$). Subcutaneous administration of 5% dextrose in water is theoretically sound, for the pool thus formed is iso-osmotic with the plasma. In fact, however, there is some redistribution of plasma electrolytes with respect to the subcutaneous pool. Some plasma electrolytes are lost to it (and this may have disastrous consequences when the electrolytes are already very low) while at the same time water (and dextrose) are being drawn into the plasma. The dextrose is metabolized, leaving a surfeit of water. With especially large and multiple clyses of dextrose-water enough of the latter may occasionally be absorbed to cause water intoxication, especially if the kidneys are diseased.

Much worse is the clysis of 5% dextrose in saline, for the subcutaneous pool will have 600 mOsm per litre. Water will be drawn out of the plasma and shock will result. This may also occur (especially in infants) when saline alone is infused subcutaneously, if the plasma concentration of electrolytes happens to be lower than that of the saline. The use of 5% dextrose-saline intravenously is reasonable (though not in infants) for the dextrose is rapidly metabolized. Adults can cope with the unusually high chloride content (154 mEq. per litre as opposed to the plasma normal of about 100 mEq. per litre) of 0.85% saline, but not infants.

Subcutaneous solutions should preferably not contain dextrose, or at the most 2.5% dextrose with hypotonic electrolytes.

## DEXTROSE, GLYCOGEN, POTASSIUM

The elimination of dextrose via the kidney (in diabetes, or the excessive administration of intravenous dextrose) also requires the excretion of water. One gramme of dextrose is equivalent to some 5.5 mM. (based on calculations involving molecular weight), so that excretion in the urine of 1 g. of dextrose requires the obligatory excretion of at least 5 c.c. water (more like 10 c.c. in young infants) and more, if extra water be available. Dehydration results. This is not due solely to the loss of water accompanying the dextrose. Somehow such osmotic diuresis (and this also applies to excess urea diuresis following on high protein feeding) interferes with the ability of the distal tubules to reabsorb sodium and water, with an excessive loss of both.[17]

Not only does dehydration result, but there is also a decrease in intracellular synthesis of glycogen together with a loss of potassium from the cells. Something like 0.36 mM. of K is deposited in tissue cells for each 1 g. of glycogen stored. Insulin plus dextrose in the treatment of diabetic coma can cause 1 mM. of K per Kg. to be transferred to the cells. This is a useful therapeutic measure not only in diabetes, but also in any state (e.g. nephritis) where the plasma K rises to dangerously high levels.

In pathological states resulting in a low plasma potassium level it is important to replace this ion and not to give Na instead, for this procedure will depress plasma potassium to even lower levels. In hypokalaemic alkalosis, such as that consequent on pyloric stenosis, renal function becomes uniquely impaired. The kidneys become incapable of excreting alkali to compensate for the metabolic alkalosis and the urine remains acid until the extracellular K be replaced. In mild potassium lack it is sufficient to infuse intravenously 2–4 mM. per D.U. Orally 10% potassium citrate provides 1 mM potassium per c.c.

## DIARRHOEA

In infantile gastro-enteritis it is common practice to use partially skimmed milk. From what has been written on kidney function it will be evident that such a practice is unphysiological.[7, 18] Darrow has stressed that the food constituents supplied should be considered in terms of calories metabolized and skimmed milk presents an unusually high osmolar load (protein) per calorie as compared to whole milk. Moreover, the fat in the milk is an appreciable source of water when catabolized, producing something like 1 c.c. of water of oxidation per g. of fat. Water of oxidation made available to the body is in the order of 10 c.c. per 100 calories metabolized, and in this respect fat is twice as efficient as carbohydrate or protein, producing twice as many calories gramme for gramme and twice as much water per gramme catabolized.

The use of half-cream milks for the normal feeding of infants in the first few weeks of life (as is commonly done in England) is also irrational. Just at the time when the infant's

kidneys are not capable of high solute concentration, they are presented with a high solute load: a high protein content per calorie supplied. This is unphysiological and may be dangerous if excessive quantities of water are lost from the body for any reason whatever.

## WHITHER CLINICAL BIOCHEMISTRY?

There is little question but that within the next decade or two simple bedside electrolyte studies on plasma and urine will become an indispensable function for proper patient care. More than 10 years ago the crude Fantus test for urinary chloride ushered in the era of bedside biochemistry. At present bedside 'do-it-yourself' kits are available for rapid and accurate determinations of serum Cl, Na, total base, bicarbonate and blood urea nitrogen,[19] and we may expect that further simplifications and refinements in this field will be made available in the future.

Such biochemical investigations will be a valuable check on clinical judgment, though no doubt biochemical interpretations will continue to be—only more so—a humbling experience. Clinical judgment has been stated to be an all-important function:

'. . . sets of rules for the parenteral administration of fluids to sick babies can never be a substitute for clinical judgment, and the riper the judgment, the less the rules are needed or heeded.'[5]

However, clinical acumen is an inexact and elusive quality. Is not Hippocrates credited with the aphorism: 'Life is short, the art long, *experience fallacious, judgment difficult.*'? Who among us can remain innocent when we consider the clinical blunders of our supposedly ripe judgment, long experience or arduous training? Judgment is subject to whims and fancies that may not be supported by objective data. When these do come to hand it is salutary to note how often the mighty are fallen.

At present, with all our uncertainties in the field of fluid and electrolytes, it is still necessary to call attention to Bakwin's plea[2] for not repeating the absurd mathematical calculations in the field of fluid therapy in the same way in which these once ruled in the realm of infant feeding. However, it is good to know of the mathematics of fluid derangements and therapy, for thereby one can use such knowledge intelligently in planning simple means for the care of our patients. A short cut to therapy is always to be welcomed, but he who would traverse a short cut with safety will be better equipped to do so if he also knows the long way round—hence the many pages of 'hexose-6-phosphate' mathematics that have preceded these concluding paragraphs.

It is also necessary to recall the presence of the kidney within the body, a remarkable structure that plays the principal role in body homeostasis:

'. . . homeostatic mechanisms are normally capable of restoring metabolic normalcy in accordance with individual needs . . . (such) mechanisms are normally more important and effective in this regard than the type of therapeutic regimen . . . it is clear that these (homeostatic) systems are ordinarily capable of accomplishing this task (recovery) with great precision when provided with raw materials at reasonably generous yet physiologically tolerable rates.'[20]

The kidney is a humble organ. Of unprepossessing size and appearance, it lies hidden within the uttermost parts of the abdomen. Far from the prying eye and curious ear this meek structure presides over the composition of the whole body. All the most secret and exquisite adjustments are performed by this arbiter of homeostasis; yet this is done with a versatility and a humility far removed from that displayed by that brash pump—the heart —or the pulmonary bellows.

## REFERENCES


1. Levin, S. (1955): S. Afr. Med. J., **29**, 1261.
2. Bakwin, H. (1953): New Eng. J. Med., **248**, 227.
3. Talbot, N. B. and Ritchie, R. H. (1959): Pediat., **24**, 495.
4. Oliver, W. J. *et al.* (1958): J. Amer. Med. Assoc., **167**, 1211.
5. Editorial (1959): Lancet, **1**, 822.
6. Darrow, D. C. (1959): Pediat. Clin. N. Amer., **6**, 29.
7. Cooke, R. E. (1959): *In* Nelson's *Textbook of Pediatrics*, 7th ed. Philadelphia: W. B. Saunders Co.
8. Wallace, W. M. (1953): Amer. J. Clin. Path., **23**, 1133.
9. Darrow, D. C. (1959): Amer. J. Dis. Child., **98**, 416.
10. Fomon, S. *et al.* (1959): Pediat., **23**, 113.
11. Smith, C. A. *et al.* (1948): Pediat., **3**, 34.
12. Pratt, E. L. *et al.* (1948): Pediat., **1**, 181.
13. Norman, F. A. and Pratt, E. L. (1958): J. Amer. Med. Assoc., **166**, 2168.
14. Holliday, M. and Egan, T. J. (1959): Pediat. Clin. N. Amer., **6**, 81.
15. Baskin, J. L. *et al.* (1952): Amer. J. Dis. Child., **84**, 351.
16. Wynn, V. (1956): Metabolism, **5**, 490.
17. Lewis, A. A. G. (1957): Proc. Roy. Soc. Med., **50**, 999.
18. Finberg, L. (1958): Pediat., **22**, 2.
19. Oski, F. A. (1959): Pediat. Clin. N. Amer., **6**, 317.
20. Talbot, N. B. *et al.* (1955): New Eng. J. Med., **252**, 856, 898.


## PRINCIPAL REFERENCES


Barness L. A., *Ed.* (1959): Pediat. Clin. N. Amer., **6**, February. Philadelphia: W. B. Saunders Co.

Hill, F. S. (1954): *Practical Fluid Therapy in Pediatrics.* Philadelphia: W. B. Saunders Co.

# FLUID LEVELS WITHIN THE PARANASAL SINUS FIELD

## THEIR NATURE, CHARACTER, BEHAVIOUR AND SIGNIFICANCE

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The radiological recognition of free fluid within the paranasal sinuses is based at present upon the conventional single-celled textbook sinal field, and the belief that all fluid levels are horizontal and remain so whichever way the head is tilted in the erect position (invariant horizontality).[1,21] As these ideas are at variance with anatomy, pathology and physical laws, it is the purpose of this paper to examine the subject anew, and to present fresh ideas and more dynamic concepts derived from the paranasal sinus fields of practice themselves.

### THE SINAL FIELDS OF PRACTICE

Since diagnosis is concerned not only with disease but also with its localization, correct anatomical knowledge is clearly essential.

Within the sinal field it is common practice to visualize and believe in frontal, maxillary, sphenoidal and ethmoidal air cells of textbook development, arranged in pairs in relation to the nasal airway, i.e. paranasally. This concept idealizes sinus anatomy and denies humanity its birthright.

It is universally acknowledged that the physiological process of pneumatization is unpredictable. Thus, development ranges from complete failure to develop (agenesis) up to the growth and encroachment of one sinus upon another not necessarily of the same group.[2]

Agenesis apart, there is variation in numbers too. In the frontal area Schaeffer[3] has observed one specimen with as many as 6 frontal sinuses. However, Davis[4] puts the frequency of 'supernumerary' cells within the frontal bone at 3–5% (Figs. 1, 2). Thus, one out of every 20–30 people has more than 2 frontal sinuses.

In the maxillary field the writer has drawn attention to 3 known varieties of 'duplicate' antrum, and added a 4th, viz. the enclosed variety.[5] Gruber[6] and Schaeffer,[3] by including the forward and downward encroaching posterior ethmoidal cells (ethmomaxillary type of duplication), put the frequency of antral duplication at 2.5%. Thus, one person in 40 has one or other variety of 'duplicate-cell' structure within one or both maxillary fields (Figs. 3, 4, 11, 12 and 31).

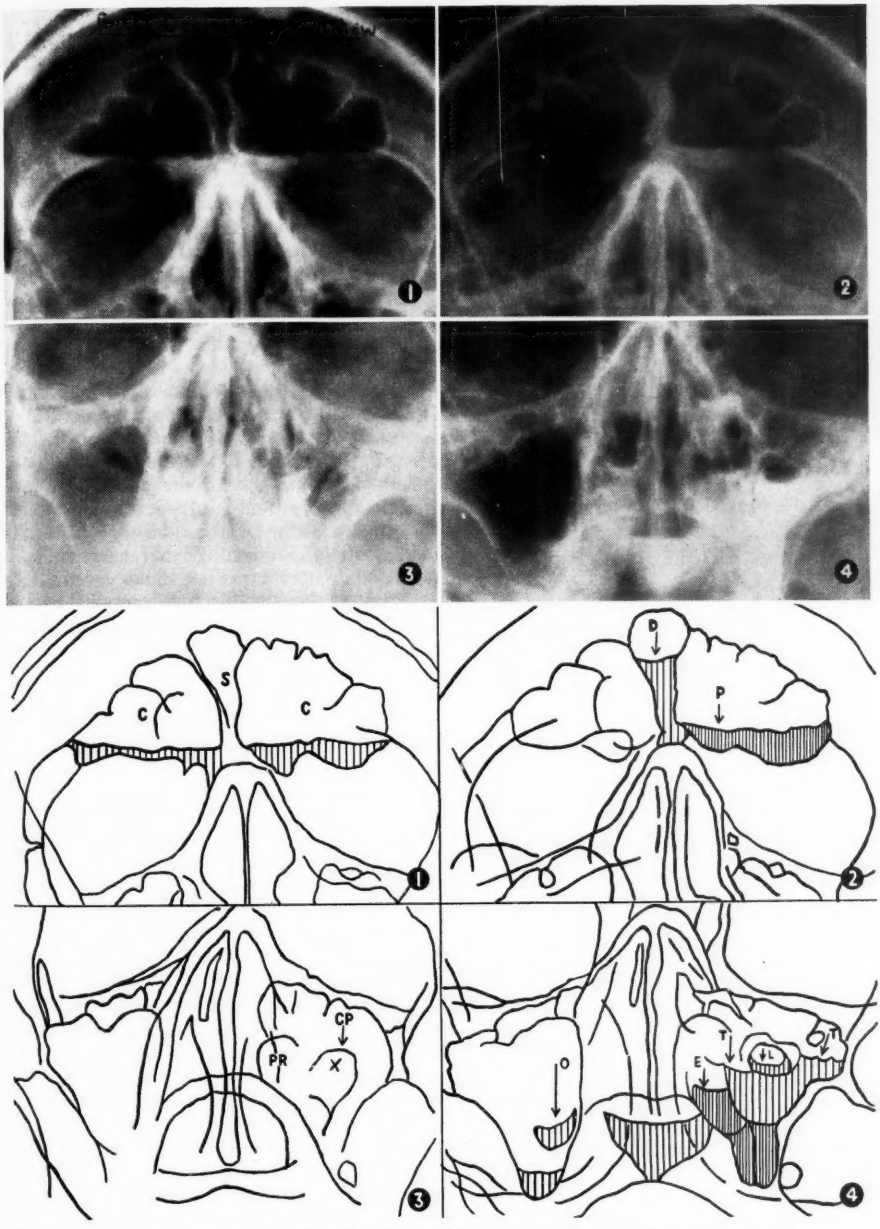
In the sphenoidal area most so-called supernumerary sphenoidal cells are really backward-encroaching posterior ethmoidal cells.[2] True supernumerary sphenoidal sinuses, however, do occur, though no figures exist regarding their frequency.[2]

In the ethmoidal area anything from 3–15 cells may be found on either side.[2]

There is no fixed arrangement and relationship between paranasal sinuses either. In the first place, intersinal septa separating conventional paired frontal and sphenoidal sinuses are rarely in the mid-line, and cells may overlap, indent, encroach upon and even surround one another. In the frontal bone cells may lie not only alongside (adjoin) but apart from, behind, and between one another (Figs. 1, 2). In the maxillary area intervening septa are also variously directed. In the genetically true variety of duplication, for instance, the intervening septum lies obliquely and semi-coronally, but in the ethmomaxillary type it may lie obliquely and semi-coronally or vertically and sagitally. In the rare third type of adjoining duplicate antrum the common bony wall is vertical and coronally directed.[8] In this variety one antrum thus lies directly behind the other within the maxilla. In the fourth variety of duplication the somewhat globular wall of the duplicate cell lies 'inside' the boundaries of the true antrum,[5] at a distance determined by the relative size of the cells (Figs. 3, 4). In the ethmoidal area the cells have strong tendencies to expand and encroach upon one another, and upon their neighbours.[7]

Nor are the paranasal sinuses always separate from each other. There may be communication between cells. In the maxillary region, for instance, communication between cells of the genetically true variety of antral duplication is by no means infrequent. This may be developmental or pathological in origin.[5] In the frontal and sphenoidal regions, however, intersinal septa are almost never incomplete.[2]

Intrasinal space itself is not a uniform or standardized entity either. Sinuses may be subdivided by ridge, septum and intrasinal pathology, e.g. cyst, polyp, lobulation, etc. Frontal sinuses, e.g. are often irregularly sub-




*Note to Illustrations:* In all cases the explanatory line drawing is keyed with the same identifying number as the half-tone illustration.

*Fig.* 1. Unfilled *supernumerary* frontal sinus (S) located *between* contrast-containing frontal sinuses (C). Note the fluid levels on the same plane.
*Fig.* 2. Supernumerary cell and left frontal sinus filled by displacement. Note fluid levels (D and P) on different planes proving triplicate cell nature of the frontal sinus field seen in *Fig.* 1.

*Fig.* 3. Note central air space (X), with calcified upper pole (CP), inside conventional antral limits. Note large palatal recess (PR).
*Fig.* 4. After displacement with watery opaque medium. Note separate markedly concave fluid level (L) in central air space (X), projecting above fluid level (T) in true antrum. Note wavelike lateral portion of antral level (T) corresponding with the intrasinal ridges and reaching to the outermost limits of the zygomatic recess, thus discounting the possibility of thickened mucosa suggested by the appearances in Fig. 3. Note fluid level (E) in palatal recess (PR) on a lower plane than other levels. The corresponding line-drawing shows the composite structure of this irregular fluid level outline. Note oblique fluid level (O) in right pterygoid recess.

divided by intrasinal ridges. Close to half the maxillary antra exhibit intrasinal projections too, ranging from minor ridges up to large crescentic partial septa productive of significant pockets difficult to drain.[3] The sphenoid cavity is also notoriously irregular and unpredictable. In roughly 20% of cases there is unilateral, and in 1% bilateral invasion of the pterygoid process in addition.[9] On occasion this results in the formation of a large pterygoid recess with a well-formed partial septum separating it from the main cavity (Fig. 5).

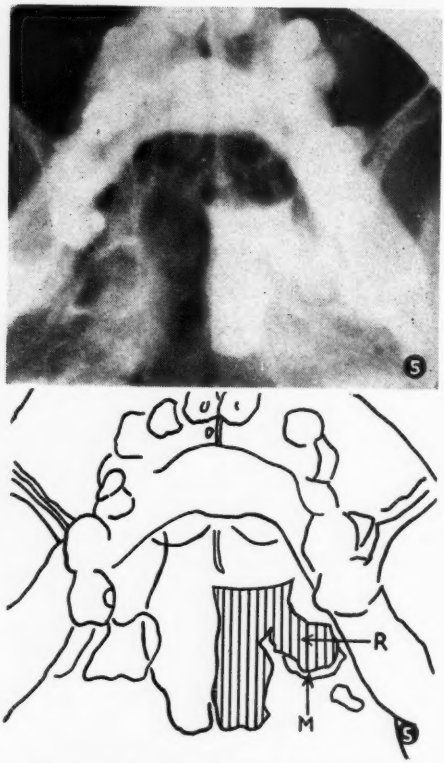



*Fig.* 5. Left sphenoidal sinus, showing large pterygoid recess (R). Filling per catheter via natural ostium with watery opaque medium in a case of migraine. Note thickened mucosa (M) in the recess.

Paranasal sinuses, varying as they do in development, numbers, arrangement, relationship, inter-communication and intrasinally, thus cannot possibly be preconceived (idealized). A concept of unvarying anatomical uniformity within the paranasal sinus field is thus arbitrary and unjustified. It leads to diagnostic ideas that make misinterpretation within the cobweb of lines and surfaces comprising the straight erect sinal X-ray film inevitable.

## THE NATURE OF THE COMPONENT FORMING THE FLUID LEVEL

A particular characteristic of some pathological sinal collections, and one favouring their radiological recognition, is the phenomenon of the fluid level. As pathological sinal collections are heterogeneous mixtures, clear ideas regarding the nature of the component responsible for this interpretive and diagnostic sign are thus of practical importance.

As no systematic treatment of the dynamics of pathological sinal collections is to be found in the literature, the writer proposes tentatively to consider this important aspect of sinal pathology. He believes it not only leads to dependable ideas concerning the component responsible for the fluid-level phenomenon, but also provides a sound theoretical basis for constructive thinking regarding the character, behaviour and significance of fluid levels and fluid-level patterns within the complex, morphologically unpredictable and pathologically variable paranasal sinus field.

Generally conceived, inflammatory sinal collections consist of exudate (plasma, tissue fluids and products of initial cellular injury),[10] fibrin, cells (white and red blood cells and tissue cells), the products of cellular disintegration, varying amounts of goblet-cell mucus and serous gland secretion, organisms, their secretions and debris. Pathological sinal collections are thus mixtures characterized mainly by elements derived from blood and sinal mucous membrane.

Wintrobe regards blood as a 'suspension of cells in plasma,' with the cells largely responsible for the 'thickness, stickiness, and viscosity' of the mixture.[11] Thus, whereas the viscosity of plasma averages 1.6–2 ($H_2O = 1$), that of normal whole blood averages 4.5.[11] When the red cells increase in numbers, as in polycythaemia, the viscosity of whole blood may become 5–8 times greater than normal, and the mixture so 'thick' as to be drawn up into a pipette with difficulty.[12] When the red cells diminish (as in pernicious anaemia, haemolytic states and profuse haemorrhage) the viscosity falls below that of normal whole blood.[13]

When red cells are the predominant element in sinal exudate, the tendency is likewise to 'thicken' it. It has been the writer's experience that haemorrhagic antral exudates form compact, semi-solid single clumps. These are

usually difficult to dislodge, and do not tend to fragment during lavage. Haemorrhagic exudates thus tend to become mechanically rigid. Any fluid that is 'free' in such circumstances will be that squeezed out during the bending and shortening of the fibrin needles in the process of forming a firmer, more rigid, elastic clot with increased resistance to stress. Red cells, fibrin and blood clots can thus hardly be responsible for the fluid level phenomenon in sinal collections.

The cells commonly predominant in pathological sinal collections are, of course, the white cells, not the red. These cells vary not only in size and number, but are present also in all stages of injury, degeneration, necrosis and liquefaction. Some idea of the influence of the size and number of white cells on the viscosity of plasma is supplied by Stephens's cases of leukemia.[14] Thus, his case of chronic lymphatic leukemia with 500,000 lymphocytes per c.mm. of cell size 6–7$\mu$, had a normal blood viscosity, whereas his case of chronic myeloid leukemia with 352,000 cells of the myeloid series per c.mm. of cell size 11–18 $\mu$ had a blood viscosity of 9.2. In the myeloid cases the blood becomes 'thick, sticky and difficult to smear' (Wintrobe). When high proportions of relatively intact neutrophil polymorphs (10–15$\mu$) are present in sinal exudates, e.g. in *Staph. aureus* infections, the mixtures become thick (porridgy) and sticky too, and difficult to shift by ciliary action, nose blowing, postnasal hawking and saline lavage. Exudates showing little evidence of clumping, on the other hand, are rarely thick and, as a rule, are easier to dislodge.

Cellular and fibrinous elements in sinal exudates thus impart thickness, sluggishness, semi-solidity, elasticity, compactness and mechanical rigidity to sinal mixtures. Clearly these elements cannot at the same time also be responsible for the fluid level phenomenon in the mixture.

It was pointed out that cellular elements in inflammatory sinal mixtures undergo injury, degeneration, necrosis and liquefaction.[15] This adds cell fluid and coarse insoluble desoxy- and ribo-nucleoproteins to the mixture. Sherry *et al.*[16] regard these proteins as the major cause of viscosity in purulent exudates. Concentrations of nucleoproteins may, however, exceed the critical value for viscous flow. They can render an exudate so glutinous that its own weight, the moving force, the flushing force of lavage, and even that of the fully opened running tap, will be insufficient to move it. Such exudates, which can stick to the sides of an inverted glass container, are no longer viscous fluids; they have become plastic masses or elastic solids.

Nucleoproteins account for some 30–70% of the sediment in purulent exudates and, according to their concentration, render it viscous, thick, lumpy, stringy, fibrous or web-like, or change it into an elastic or plastic mass. Since nucleoproteins impart varying degrees of viscosity and various patterns and forms of structural rigidity to sinal mixtures, they clearly impede and prevent flow rather than favour or create it. To imagine, let alone accept, nucleoproteins as being responsible for the fluid level phenomenon in sinal mixtures is thus not merely difficult; it is impossible.

Goblet cell mucin and diffused bacterial mucoids[17] also form part of inflammatory sinal collections. These, with their high surface tension, encourage the formation of globules. These tend to sediment to the sinal floor and change form very slowly, if at all, on tilting.[9] It is not possible to regard these elements as being responsible for the fluid level phenomenon in sinal fields either.

Clinically, antra have been seen containing gels. These are usually single, variable-sized, globular, semi-solid masses of considerable mechanical rigidity. As they are hydrophilic and act as quasi-solids, it is likewise impossible

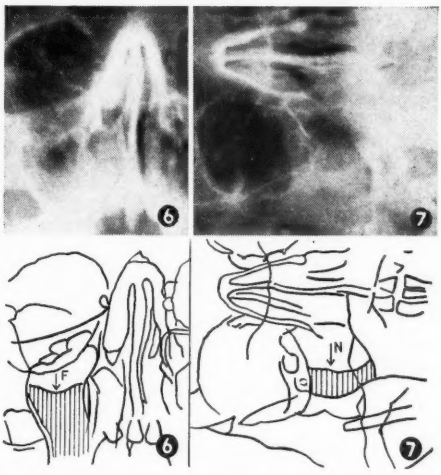
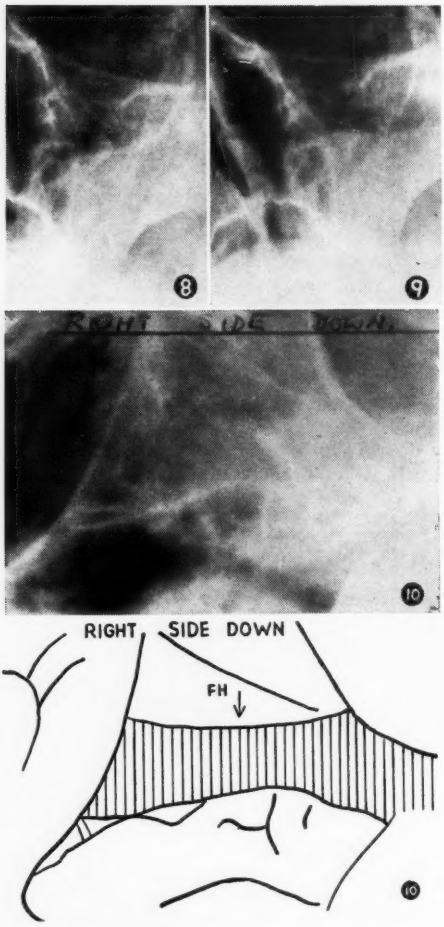



*Fig.* 6. Occipito-mental view showing horizontal level (F) in right antrum. The patient complained of milky fluid running from right nostril on stooping or bending forward.

*Fig.* 7. Right recumbent occipito-mental view, showing horizontal concave level (N) conforming to new cross-section in different erect right-angled plane, proving free fluid.

to visualize gels as being responsible for the fluid level phenomenon in sinal fields.

Organisms, too, form part of inflammatory sinal mixtures. While not in themselves physically capable of forming fluid levels, organisms do, nevertheless, indirectly assist in their formation. Initially the fluid element of inflammatory sinal mixtures consists of exudate and serous gland secretions poured out in response to the irritant. In the course of the pathological process, organisms and their toxins, and changes in H-ion concentration,[15] cause injury, degeneration, necrosis and liquefaction of intact cells, adding cell fluid to the collection. Depending on the nature of the infection, and the pathological response, extracellular enzymes are simultaneously liquefying dead, dying and degenerate cell nuclei, nucleoprotein and fibrinous matrices and scaffoldings, and viscous mucins, mucoids and intercellular cement substance, liberating and adding to the collection trapped, bound and dispersed fluids from clumps, clots, gels, globules, nucleoprotein structural forms, and coarse slimy sediments.



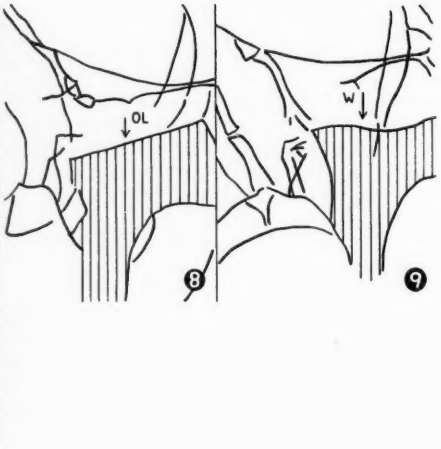




*Fig.* 8. Shadow with oblique upper level (OL) occupying lower portion of left antrum.

*Figs.* 9 *and* 10. Tilt-to-the-right and right recumbent occipito-mental positions producing undulant horizontal level (W) in Fig. 9, and a flat horizontal level (FH) in Fig. 10, proving the fluid nature of the oblique level (OL) seen in Fig. 8.

Basically, then, all inflammatory sinal collections are mixtures of varying amounts of free fluid (exudate + serous gland secretion + cell fluid + enzymatically liberated fluid) and other elements (cells + clumps + clots + strands + globules + nucleoprotein structural forms). Naturally the amount of free fluid in the mixture will influence its character, behaviour and phenomena. For instance, inflammatory sinal

collections rich in cells and slimes and poor in lytic enzymes, e.g. *Staph. aureus* infections, have masses of healthy, injured and degenerate cells enmeshed in rope-like matrices of coarse, sticky nucleoprotein. These form thick, porridgy mixtures which drain with difficulty, and are difficult to dislodge by patient and doctor alike. Radiologically such sinuses are usually cloudy or opaque, or show reduced air space from lesser combinations of mucosa swelling and adhering exudates within the cells.

Clinical reports of thin, milky fluid running or dripping from the nose on stooping or bending forward indicate, on the other hand, sinal collections with measurable amounts of free fluid within the mixture. Experiences of this kind, in cases with radiologically verified fluid levels (Figs. 6, 7), are comparable with like experiences following radiodiagnostic or therapeutic displacement with watery opaque media or with saline in sinal fields.

As only the free-fluid component of inflammatory sinal mixtures can behave uniformly and naturally in a manner identical with that *in vivo* of the 2 known free fluids 'saline' and 'watery opaque medium,' it follows that only this free fluid, and no other element, is responsible for the fluid level phenomenon in inflammatory collections within the paranasal sinus field.

## THE CHARACTER AND BEHAVIOUR OF SINAL FLUID LEVELS

It may at first sight appear that an unnecessary to-do has been made regarding the nature of the component that is responsible for the fluid level phenomenon in sinal fields. The situation facing the radiologist, however, is by no means simple. On the one hand, he is faced with paranasal sinuses that vary in development, numbers, arrangement, relationship, in-

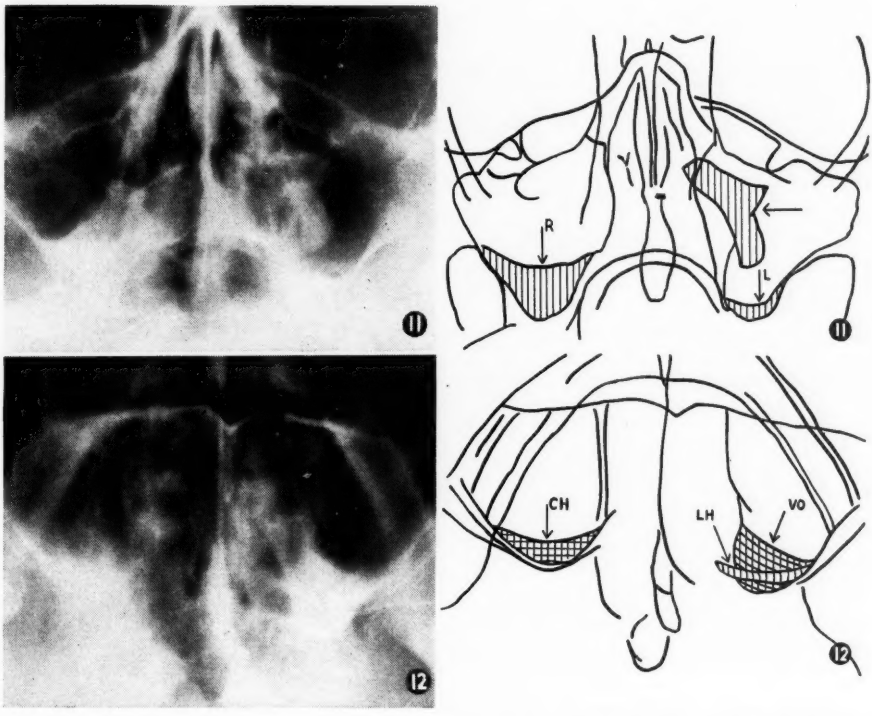


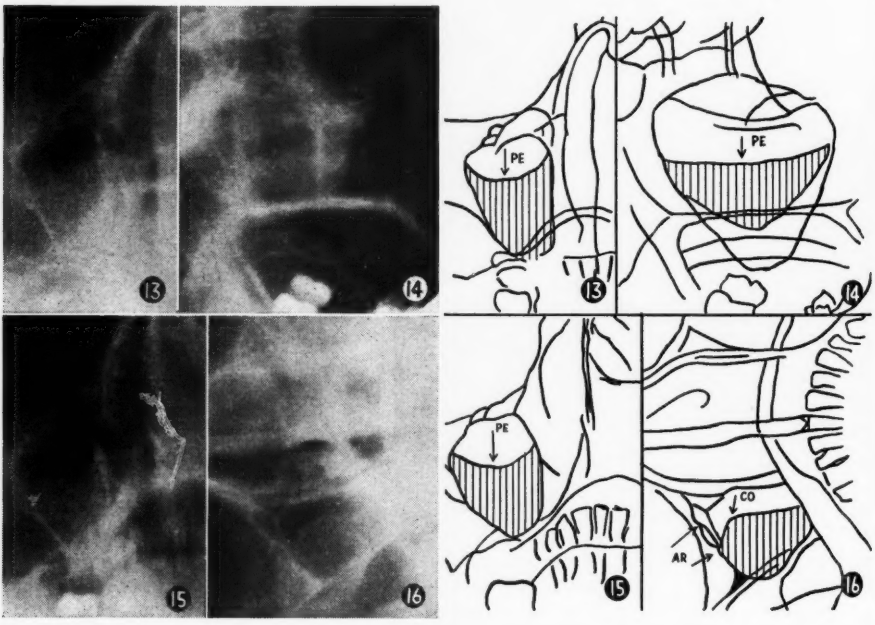

*Fig.* 11. Note horizontal opaque fluid levels (R and L) on floor of right and left antra, and irregularly shaped opaque fluid shadow (arrow) in medial centre of left antral field.

*Fig.* 12. Submento-vertical view. Note concave horizontal fluid level (CH) in the most dependent part of the right antrum, and upper oblique (VO) and lower horizontal concave level (LH) in left antral field, demonstrating failure to reproduce horizontality in left antral field on tilt testing.

tercommunication and pathology and, on the other, with components of inflammatory sinal collections that are similar in radiodensity to each other and to the contiguous and overlying sinal soft tissues (mucosa, cyst, polyp, etc.). The criteria, ideas and methods employed by the radiologist to surmount these difficulties are thus of practical importance.

About 1926 Claus[18] proposed recognition of inflammatory sinal collections on the basis that 'serous fluids were concave, mucus or muco-pus convex.' Claus was aware, however, of a weakness in these criteria. He noted that a polypus also had a convex outline. There were, however, more serious objections to Claus's criteria. In the first place, all sinal fluid levels, whether due to pathology, saline or watery opaque media, were naturally concave, and the serous and saline ones radiologically indistinguishable. In the second place, not only mucus, muco-pus and polyps, but also gels, blood clots, cysts, new growths and hyperplastic mucosa presented convex outlines. Thus concavity and convexity and the nature of the sinal shadow were not in themselves interpretively (diagnostically) interchangeable, and convexity was more a sign of solidity than fluidity. Claus' criteria, and method, thus amounted to nothing more than a description of physical characteristics having reference to nothing but themselves. His ideas took no notice of anatomy, paid lip service to pathology, and gave a mere nod of recognition to capillary action and surface tension within the sinal field. Claus was thus an 'archivist of facts' (Pavlov), not the discoverer of a diagnostic law. Mere description is not diagnosis.

In 1946 Pillmore[19] asserted that 'serous and irrigation fluids gave clear-cut fluid levels, pus a meniscus-like level with curved outer margin less clear than the serous or irrigation fluid.' As pus, muco-pus, blood, saline irrigating fluid,[20] serum and contiguous and overlying sinal soft tissues are radiologically indistinguishable, Pillmore's criteria were in essence nothing more than Pillmore's own personal beliefs. His ideas made no mention of anatomy, overlooked pathology and gave but scant



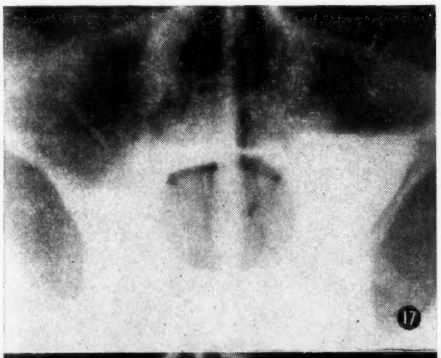

*Figs.* 13 *and* 14. Note horizontal level (PE) in erect occipito-mental and lateral views; presumptive evidence of free fluid.

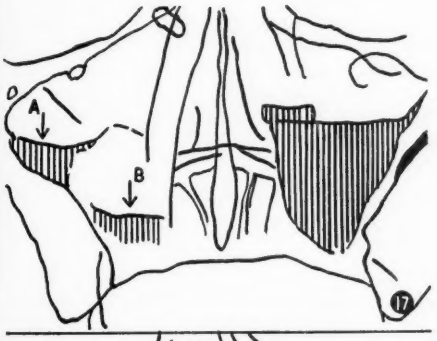
*Fig.* 15. Reproduction of horizontality (PE) on partial-tilt testing, apparently showing presence of free fluid in antrum.

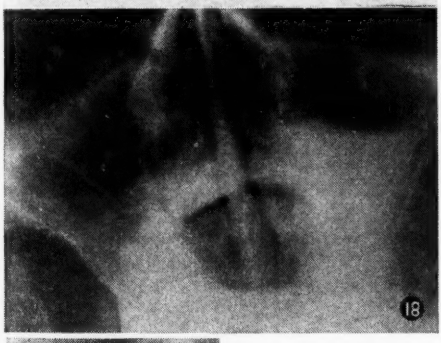
*Fig.* 16. Right recumbent occipito-mental view showing shadow in Figs. 13-15 with convex upper outline (CO) falling well short of antral roof (AR), demonstrating a fixed, non-tense, non-secreting cyst anchored to the antral floor and not free fluid.

recognition to capillary action within the sinal field. Since ' the kernel of the scientific outlook is the refusal to regard our own desires, tastes and interests as affording a key to the understanding of the world ' (Bertrand Russell), it was not surprising that Pillmore's eclectic method for recognizing the free-fluid component of inflammatory sinal collections soon proved itself unequal to its diagnostic responsibilities.

At present, recognition of the free-fluid component is based upon the belief that all sinal fluid levels are horizontal, and remain so whichever way the head is tilted in the erect position.[1,21] This standard, rule and test, however, does not accord with experience either. In the first place, not all sinal fluid levels are horizontal (Figs. 8–10),[1] nor is horizontality always reproducible on tilting (Figs. 11, 12).[1] In the second place, a non-tense sinus cyst (a non-free element) can present a horizontal upper limit which the customary partial tilt test can reproduce (Figs. 13–16).[1] Thirdly, it is not possible to limit the behaviour of a truly ' free ' fluid to the reproduction and visible demonstration of horizontality within unpredictable and variable sinal space. Far from remaining visible and horizontal on tilting, free fluid may disappear





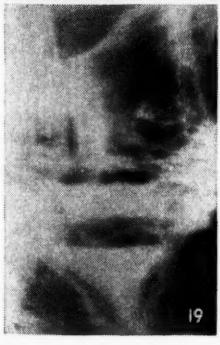




*Fig.* 17. Note 2 opaque horizontal fluid levels (A and B) in the right antrum, one (A) extending from the apex of the antral cyst to the zygomatic recess, and the other (B) occupying the inferior angle of the antrum.

*Figs.* 18 *and* 19. The tilt-to-the-right view (Fig. 18) shows both fluid levels are still separate and horizontal.

The left recumbent occipito-mental view (Fig. 19) shows both fluid levels have *disappeared*, presumably behind the cyst, demonstrating the character of a level is not always 'visible', or 'horizontal' (see Fig. 12), on tilt-testing.

behind swollen mucosa, polyp or neoplasm, or into or behind a pathologically involved 'supernumerary' cell, sinus or compartment, here to disgrace itself still further by forming a non-horizontal fluid level (Figs. 11, 12 and 17–19).

Presently held diagnostic ideas for the recognition of free fluid in sinal fields thus overlook the variability of sinal space (anatomical and pathological) and the laws of capillarity and, on the basis of the customary partial-tilt technique of verification, fail to provide unequivocal proof in all instances of the nature and free-ness of the X-ray sinal shadow. The standard, rule and test of invariant horizontality is thus based upon an assumption. Since theory must reflect Nature, and not Nature theory, it must be obvious that this method of diagnosis, based upon the expectations (preconceived ideas) of the radiologist, cannot be equal to its interpretive and diagnostic responsibilities either, within the unpredictable and variable paranasal sinus field.

## THE CONCEPT OF CO-VARIANCY FOR THE RECOGNITION OF FREE FLUID

Experience shows that descriptive methods (vision), eclectic notions (preference) and preconceived ideas (bias) are theoretically and methodologically inadequate for the proper and accurate recognition of free fluid in sinal fields. Since 'theory is man-made, and truth that which stands the test of experience, science seeks relations which are thought to exist independently of the searching individual, including the case where man himself is the subject' (Einstein).[22]

Interpretive ideas for the recognition of free fluid in sinal fields must thus take into account anatomy, pathology, capillary laws and the essential quality of 'free-ness' of the fluid. The writer is persuaded that these interpretive and diagnostic requirements are met by his concept of co-variancy, which he derives as follows:

The behaviour of all sinal fluid levels, whether of pathological collections or saline or watery opaque media, is dependent on the same basic mechanisms. Since pathological and saline fluids, and contiguous and overlying sinal soft tissues (mucosa, polyp, cyst, etc.) are radiologically indistinguishable, watery opaque fluids make it clear that all sinal fluid levels are, in fact, concave in character (Figs. 20, 21).

All sinal fluid levels denote the meeting place between fluid and air (atmosphere).[23] As waves, such as occur on wind-swept open water, cannot occur on fluid pools within the confined, protected paranasal air spaces, all sinal fluid levels (menisci, concave variants) thus betoken wetting and spread on 'solid' (non-aerial) limiting surfaces.

Thus, all sinal fluid levels are concave, and connected with, dependent on, and determined by the degrees of separation between the limiting surfaces of the zones of pooling and collection within an unpredictable and variable sinal field. Since the degree of concavity is an inverse function of the degree of separation, and capillarity influences its height without altering its basic character, it follows that 'capillary manifestations in concave variants' is the correct description and prediction (generalization) of the character and behaviour of a sinal fluid level, and that these fluid levels are co-variant with the characteristics of space at the level of sinal pooling in all erect posi-

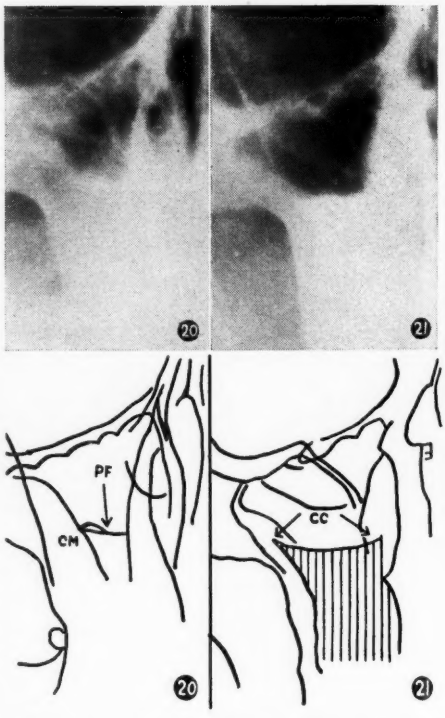



*Fig.* 20. Note impossibility of differentiating limits of pathological free fluid level (PF) from contiguous swollen mucosa (CM).

*Fig.* 21. Opaque fluid level demonstrating the concave character (CC) of free fluid levels (menisci) in paranasal sinuses.

tions of the skull. Since a fluid level is thus not only a pathological sign but an index of space and dimension, it follows that only shadows capable of forming concave variants (menisci, fluid levels) in 3 erect planes (films) at right angles to each other can truthfully be interpreted as free fluid in sinal X-ray films (Figs. 11, 12, 31, and 13–16).

Thus, in terms of theory as well as experience, the character and behaviour of a free fluid level is co-variant with the characteristics of space at all levels of sinal pooling in all erect positions of the skull. It follows, then, that a concept of 'invariant horizontality' can thus have no place in the radiodiagnosis of free fluid within the unpredictable and variable paranasal sinus field. Like the concepts of Claus and Pillmore, it too belongs to history.

## FLUID-LEVEL PATTERNS AND THEIR SIGNIFICANCE IN SINAL FIELDS

It is common radiological practice to 'believe in' conventional single-celled sinal fields (frontal, maxillary, sphenoidal) and single horizontal fluid levels, with the latter, presumably, also signifying one cell, sinus or compartment.

It has, however, been pointed out that paranasal sinuses vary in development, numbers, arrangement, relationship and intercommunication, are subject to subdivision by ridge, septum and intrasinal pathology, separation by variously directed partitions, given to recess formation, overlapping, indentation and encroachment, and to lying alongside, behind and within one another.

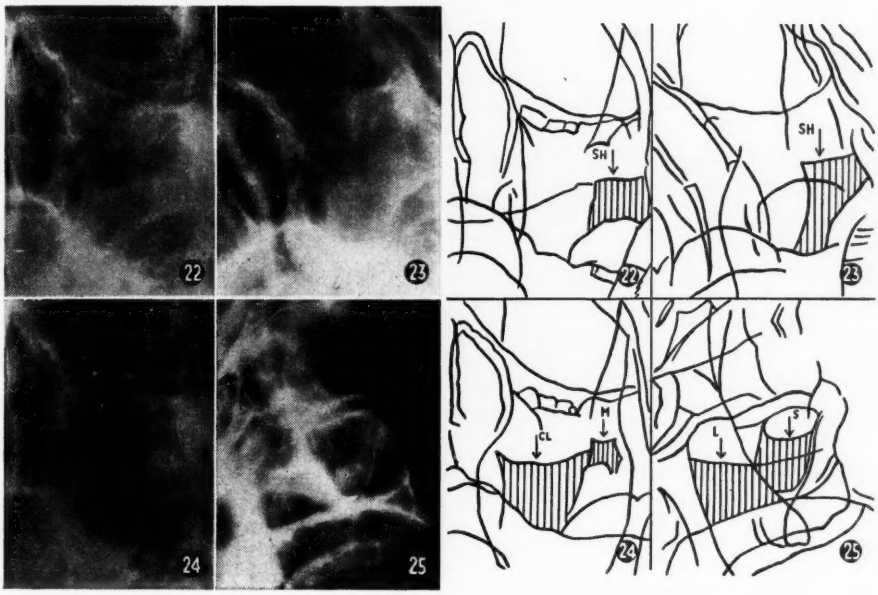



*Fig.* 22. Note short, flat horizontal level (SH) in the left antrum, extending from the zygomatic recess for for a short distance medially well short of the medial wall of the true antrum, and projecting slightly proximal to the medial convex portion of the shadow outline. Presumptive evidence of pooling of free fluid.

*Fig.* 23. Tilt-to-right suggests free fluid nature of level (SH) seen in Fig. 22. Note that the medial convex portion of the shadow outline is unchanged in position and appearance.

*Fig.* 24. Reproduction of appearances in Fig. 22, following displacement with a watery opaque medium (M). Note *conventional* fluid level (CL) on a slightly lower plane stopping short at the medial boundary of the medium (M).

*Fig.* 25. Erect left lateral view, showing 2 separate fluid levels (S and L), one (L) of 'conventional' limits, the other (S) proximal to its anterior portion and well short of conventional limits. A case of suppuration in a non-communicating duplicate antral field. Two-catheter test positive[6].

Since free fluids collect, pool and form fluid levels in air cells of differing development, numbers, arrangement, relationship, intercommunication and pathology, fluid levels in sinal fields can vary not only in character, but also in extent, number, position, arrangement and relationship. Anatomy, pathology and physical laws, and not preconceived ideas, thus determine the patterns and significance of the behaviour of free fluid within the sinal fields of practice.

From the standpoint of practical radiological diagnosis free fluid must thus be recognized not only when but also where it occurs. As free and encysted fluid, clumps, clots, gels and polypoid, oedematous and swollen mucosa are similar in radiodensity, it is logical and realistic to regard all sinal shadows with horizontal, oblique or irregular upper borders, whether of conventional limits, occurring within or falling short of them, as presumptive evidence of pooling and collection requiring 'tilt-testing' (Figs. 22–25).

Fluids that collect, pool and form fluid levels, however, obey physical laws, and pools and levels form patterns capable of analysis and interpretation in sinal X-ray films. There occur, for instance, wave-like patterns, a series of fluid levels (menisci) linked together on the same plane within the field (Figs. 26, 27).

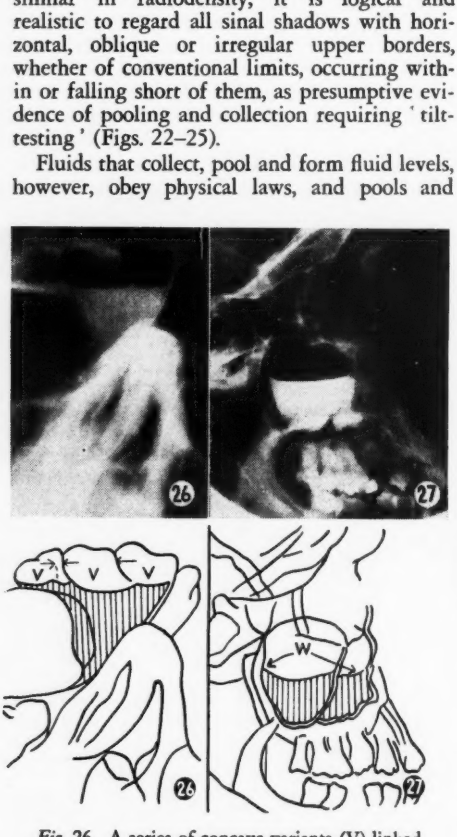



*Fig.* 26. A series of concave variants (V) linked together on the same plane. Wave-like pattern. Points of linkage correspond to intrasinal ridges in the frontal sinus (arrows).

*Fig.* 27. Erect lateral view, showing wave-like pattern (W) in antral field and suggesting the possibility of communicating duplicate cell structure.

Primarily, of course, such a pattern betokens subdivided space. As, however, the radiological invisibility of wall, septum and partition does not alter structure and physical laws, the possibility exists that the pattern may betoken multiple cells communicating below fluid level. Since such structure may be responsible for inadequate drainage and unsuccessful lavage in chronically infected sinal fields, the possible significance of this pattern should therefore not be overlooked. Complete right-angle tilt testing will be of assistance in deciding whether a communicating duplicate-cell field is responsible in particular instances (Figs. 27–30).

There are, of course, sinal fields where multiple cells do not communicate with one another, and these cells may overlap, indent, encroach upon or lie behind, alongside, or within one another (Figs. 1–3, 11, 12, 24, 25). Naturally, more than one fluid pool can occur in such sinal fields. Single, or separate shadows with fluid-like levels of unconventional extent and situation (Figs. 11, 12, 22, 23), as well as shadows with irregular outlines made up of adjoining levels (menisci) on different planes, can thus represent patterns of pooling and collection in non-communicating multiple-cell sinal fields (Figs. 1, 2).

As proof puncture rarely, if ever, includes the posteriorly located cell in a duplicate antral field,[5, 24] and as 'frontal' sinusitis may be due to involvement of a centrally or posteriorly located cell within a multi-celled frontal sinus field (Figs. 32, 33), films in two different planes together with right-angle tilt testing will generally be necessary for the correct analysis, interpretation and localization of such sinal shadows.

However, not all fluid shadows with irregular outlines made up of adjoining levels on different planes are due to non-communicating cells within the field. In some instances duplicate cells communicating high up on a common wall are responsible for this pattern. Such a mechanism was operative in an infected duplicate antral field that was filled in the ipsi-lateral recumbent position following catheterization via a single ostium. X-ray in the erect lateral position showed 2 adjoining opaque fluid pools with levels on different planes, the one in the posterior compartment being at the higher level (Fig. 34). In other

instances incomplete fusion of a double maxillary pouch may be responsible for the pattern. In these cases variously directed residual septa are responsible for pockets of differing cross-section. In the presence of free fluid, adjoining levels on different planes are brought about by capillary action (Figs. 35, 36).

Finally, vagaries of pneumatization and pathology together can be responsible. In shallow cells like the frontal sinus, for instance, the apposition of swollen mucosa on intrasinal ridges and opposite sinal wall (*cf.* occluded ostium) sometimes results in tube-like spaces. In the presence of free fluid capillary action may be responsible for adjoining levels on different planes (Figs. 37, 38).

By the demonstration *in vivo* of oblique levels (Figs. 4, 8, 12), multiple levels (Figs. 2, 4, 28, 34, 37), undulant levels (Fig. 9), irregular levels (Figs. 35, 37), linked levels (Figs. 26, 27, 37), composite levels (Figs. 4, 37), levels on the same plane (Figs. 26, 27) and on different planes (Figs. 2, 4, 24, 28, 31, 34, 35, 37), and levels up to (Figs. 8–10, 17) and short of conventional limits (Figs. 4, 23, 31, 32), in addition to the single horizontal variety (Figs. 1, 6, 7, 10), it becomes clear that fluid-level behaviour, the patient's structure (anatomical and pathological) and physical laws are indivisible, interdependent and interrelated.

Diagnostic ideas based upon predetermined conditions and one variety of fluid-level behaviour within the unpredictable and variable paranasal sinus field thus represent arbitrary static concepts, which are biased and unrealistic, interpretively inadequate, and fallible.

## CONCLUSION

Since idealistic conceptions of a fixed, unchanging order, uniformity and regularity in regard to fluid-level behaviour and sinus anatomy are contrary to nature, contrary to experience, and rejected by experiment *in vivo* with opaque fluid media, it follows that the twin concepts of 'invariant horizontality' and the conventional single-celled textbook sinal field are unequal to their interpretive and diagnostic responsibilities in practice.

Concepts that do not interpret Nature, only imagine, censor and overlook her, are patently inimical to diagnosis and should be discarded. Evidence shows that sinal fluid levels are, first

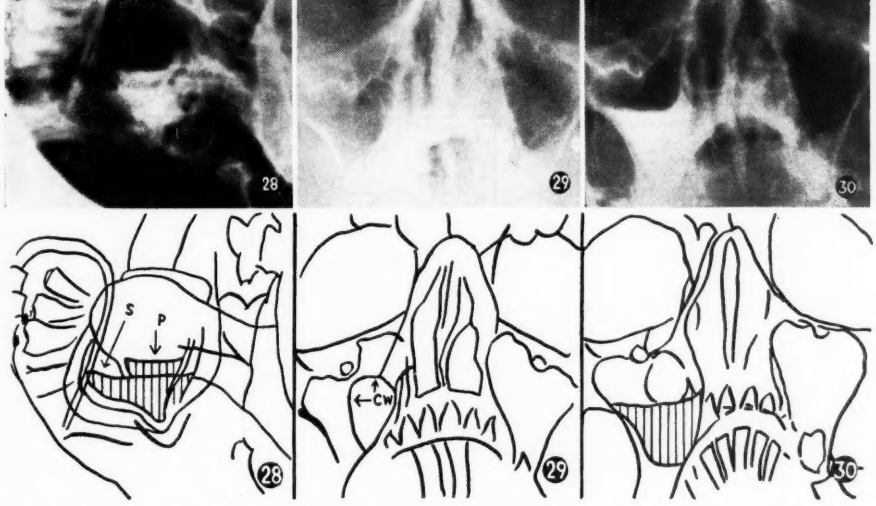



*Fig.* 28. Prone lateral view, demonstrating 2 separate fluid levels (S and P) on different planes and the duplicate cell structure of the field.

*Fig.* 29. Note well calcified duplicate cell wall (CW) in centre of right antral field.

*Fig.* 30. Note how fluid shadow can obscure the structure of a field, as shown by Figs. 27 and 28.

and foremost, 'free' fluids collecting, pooling, and obeying physical laws in morphologically unpredictable and pathologically variable para-nasal sinus fields. A sinal fluid level is thu not just simply an invariantly horizontal sig having reference to nothing but its patho

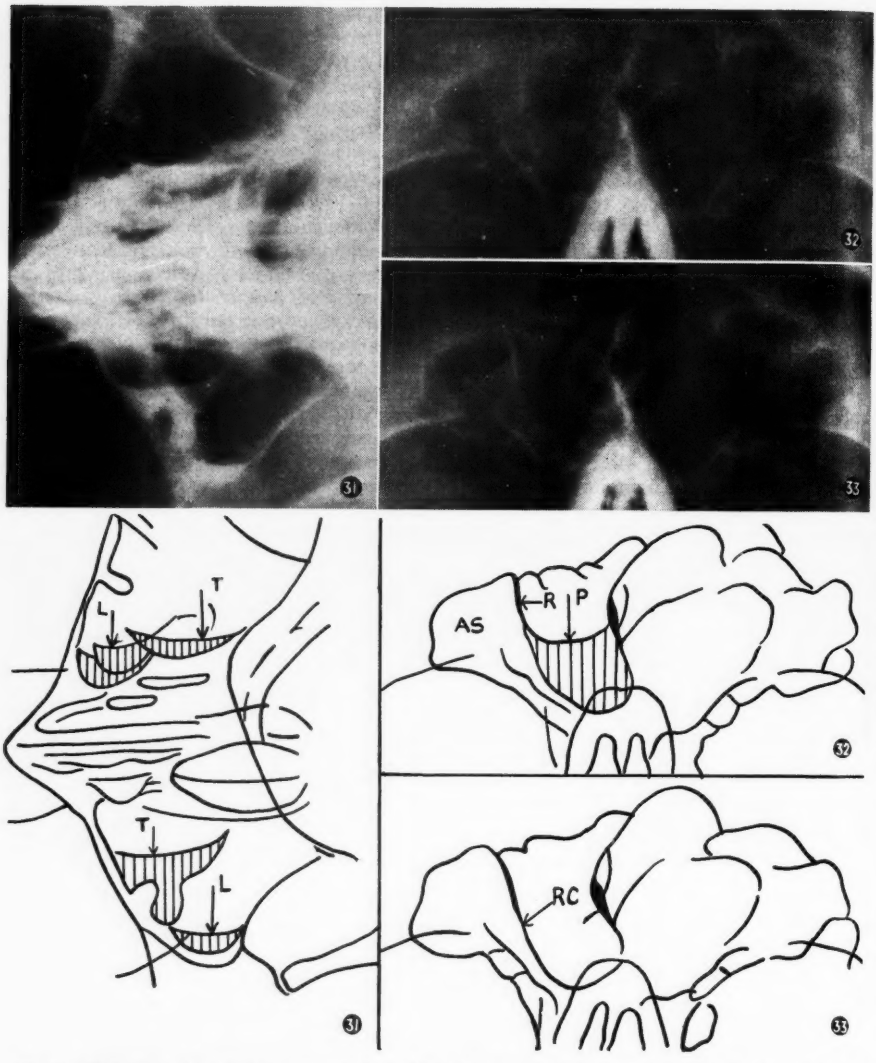



*Fig.* 31. Right recumbent occipito-mental view. Note 2 separate fluid levels (T and L) on different planes in both antral fields. Note that uppermost level (T) in right antral field is *unconventional* in length and situation. This view, with Figs. 11-12, demonstrates bilateral non-communicating antral duplication in a case of sinusitis, and illustrates the fundamental principle regarding 'proof' of free fluid in sinal fields. *Cf.* Figs. 13-16.

*Fig.* 32. Pathological fluid level (P) in 'middle cell' of triplicate frontal sinus field. Note lateral limit of level corresponding to ridge-like wall (R) and air-space (AS) of outermost cell to the right of it. The right and left recumbent views proving triplication were unsatisfactory for reproduction. A case of a colleague complaining of a 'right-sided' frontal sinusitis.

*Fig.* 33. Following cure. Note returning calcification (RC) of lateral wall of central cell.

logical self, but a phenomenon co-variant with sinal space and dimension, a spatial analyst, guide to structure, communication, and morphology, as well as guide to diagnosis, treatment and prognosis. Such a concept, neither arbitrary nor static but living and dynamic, not only expresses the totality and significance of the phenomenon within the field in question, it frees the 'observer' from the mental bonds of preconceived ideas and supplies him with the true interpretive and analytical elements of fluid level and structural behaviour in practice.

## SUMMARY

1. At present the twin concepts of 'invariant horizontality' and the conventional single-celled textbook sinal field constitute the foundations of free fluid and structural diagnosis within the paranasal sinus field.

2. Attention is drawn to the fact that sinal fields are morphologically unpredictable and pathologically variable, and intrasinal space not a fixed, uniform, immutable entity.

3. The dynamics of inflammatory sinal collections are considered, and a truly 'free' fluid component is shown to be the element responsible for the fluid level phenomenon in sinal fields.

4. The various ideas advanced from time to time for the recognition of free fluid in sinal fields are considered, and their fallacies and inadequacies pointed out.

5. Fresh concepts of fluid level behaviour and free fluid diagnosis are presented, and their derivation outlined.

6. It is shown that fluid levels obey anatomy, pathology and physical laws, and not preconceived ideas, and that variations have interpretive, analytical and diagnostic significance in paranasal sinus fields.

It is concluded that presently held diagnostic concepts are unequal to their interpretive responsibilities and should be discarded.

The writer wishes to thank Drs. A. A. Meyer, E. van der Burgh, K. Gunn and S. J. Sarif for their radiological co-operation and assistance.

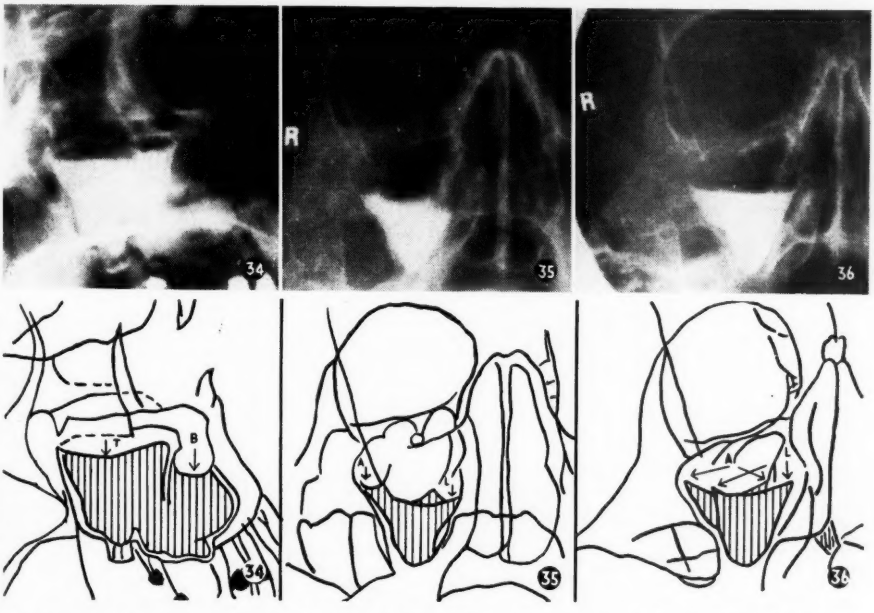



*Fig.* 34. Erect lateral view, showing 2 fluid levels (T and B) on different planes following filling by catheter in ipsilateral recumbent position via a single ostium. Illustrates posibility of
(i) Communication high up on common wall in duplicate-cell field, and
(ii) Fluid shadow with irregular step-like outline in communicating duplicate cell field.

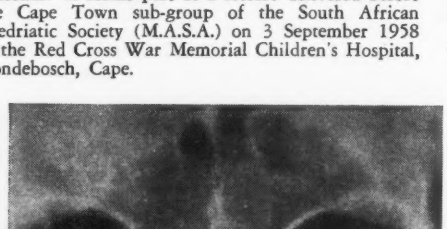
*Fig.* 35. Adjoining levels (A and L) on different planes producing irregular fluid level outline. Fluid introduced via 2 separate ostia.

*Fig.* 36. A change of tilt altering levels of pooling seen in Fig. 35. Note 2 fluid levels of different extent now on the same plane. Illustrates possibility of irregular fluid level outline in loculated single cavity by capillary action.

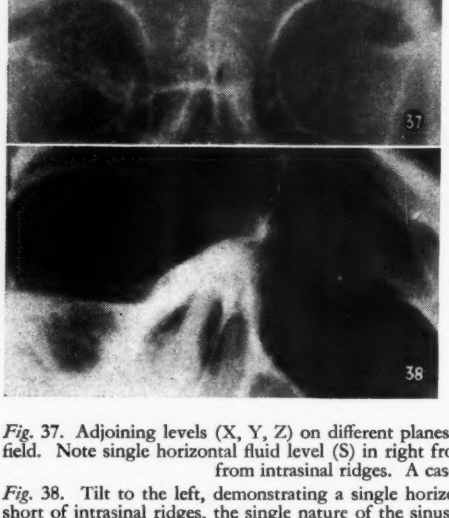
Special thanks are due Drs. E. van der Burgh and J. N. Jacobson, whose personal association, interest and criticisms have been invaluable throughout, and to Mr. S. J. Rushworth, who was responsible for most of the radiographical work.

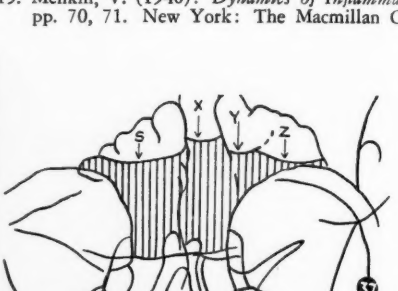
Thanks are also due Prof. J. H. Louw, of the Department of Surgery, University of Cape Town, for making available the photographic services of Mr. G. McManus, to whom thanks are also due.

This paper is dedicated with respect and affection to my teachers Prof. C. F. M. Saint, Prof. E. C. Crichton, Prof. F. Forman and the late Prof. A. W. Falconer. It forms part of a lecture delivered before the Cape Town sub-group of the South African Paedriatic Society (M.A.S.A.) on 3 September 1958 at the Red Cross War Memorial Children's Hospital, Rondebosch, Cape.





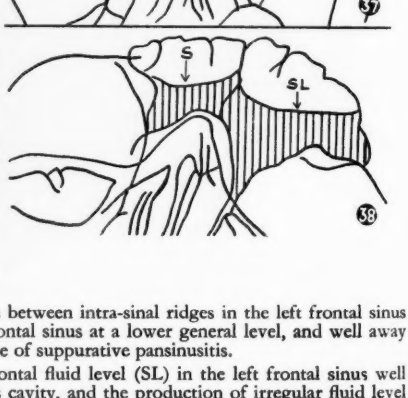




*Fig.* 37. Adjoining levels (X, Y, Z) on different planes between intra-sinal ridges in the left frontal sinus field. Note single horizontal fluid level (S) in right frontal sinus at a lower general level, and well away from intrasinal ridges. A case of suppurative pansinusitis.

*Fig.* 38. Tilt to the left, demonstrating a single horizontal fluid level (SL) in the left frontal sinus well short of intrasinal ridges, the single nature of the sinus cavity, and the production of irregular fluid level outlines in the subdivided space by capillary action, as seen in Fig. 37.

## REFERENCES

1. Glass, M. (1957): S. Afr. Med. J., **31**, 876.
2. Jackson C. and Jackson, C. L. (1946): *Diseases of the Nose, Throat and Ear*, pp. 12–15. Philadelphia and London: W. B. Saunders Co.
3. Schaeffer, J. P. (1920): *The Nose, Paranasal Sinuses, Nasolacrimal Passageways and Olfactory Organs in Man.* Philadelphia: P. Blakiston, Sons & Co.
4. Davis, W. B.: *Quoted by* Jackson and Jackson, *op. cit.*[2]
5. Glass, M. (1952): S. Afr. Med. J., **26**, 895.
6. Gruber: *Quoted by* Schaeffer, *op cit.*[3]
7. Van Alyea, O. E. (1942): *Nasal Sinuses: An Anatomical and Clinical Consideration*, pp. 48, 112. Baltimore: Williams and Wilkins Co.
8. Simon, E. (1939): Ann. Otol. (St. Louis), **49**, 640.
9. Proetz, A. W. (1931): *The Displacement Method of Sinus Diagnosis and Treatment*, pp. 108, 35. St. Louis: Annals Publishing Co.
10. Menkin, V. (1950): *Newer Concepts of Inflammation*, p. 4. Springfield, Ill.: Charles C. Thomas.
11. Wintrobe, M. M. (1946): *Clinical Hematology*, 2nd ed., pp. 225, 226. London: Henry Kimpton.
12. Wintrobe, M. M. (1946): *Ibid.*, p. 595.
13. Thorpe, W. V. (1955): *Biochemistry for Medical Students*, 6th ed., p. 166. London: J. & A. Churchill Ltd.
14. Stephens, D. J. (1936): Proc. Soc. Exp. Biol. (N.Y.), **35**, 251.
15. Menkin, V. (1940): *Dynamics of Inflammation*, pp. 70, 71. New York: The Macmillan Co.
16. Sherry, S., Tillett, W. S. and Christensen, L. R. (1948): Proc. Soc. Exp. Biol. (N.Y.), **68**, 179.
17. Dubos, Rene J. (1949): *The Bacterial Cell*, p. 37. Cambridge, Mass.: Harvard University Press.
18. Claus, G. (1926): Fortschr. Röntgenstr., **34**, 262.
19. Pillmore, G. U. (1946): *Clinical Radiology*. Philadelphia: P. A. Davis & Co.
20. Samuel, E. (1952): *Clinical Radiology of the Ear, Nose and Throat*, p. 75. London: H. K. Lewis & Co.
21. Samuel, E. (1952): *Ibid.*, p. 76.
22. Einstein, A. (1950): *Out of My Later Years*. New York: The Philosophical Library.
23. Samuel, E. (1952): *Op. cit.*,[20] p. 75.
24. Van Alyea, O. E.: *Op. cit.*,[7] p. 48.

# NOTES AND NEWS : BERIGTE

## BROXIL

Petersen Limited (P.O. Box 5785, Johannesburg; *Telephone*: 22-8911) announce that stocks of *Broxil* penicillin (BRL 152) have now arrived. Supplies are available to the medical profession through the usual pharmaceutical trade channels.

* * *

## STAPHCILLIN

B.L. Pharmaceuticals (Pty.) Ltd. (P.O. Box 2515, Johannesburg; *Telephone*: 22-2735) announce that stocks of their new penicillin (*Staphcillin*) are now available through the usual trade channels.

* * *

## BOOTS APPOINTMENT DIARY FOR 1961

Copies of this diary are available for distribution to medical practitioners. Those interested should write without delay to: B.P.D. (S.A.) (Pty.) Ltd., P.O. Box 45, Jeppestown, Transvaal.

* * *

Dr. Graham Smit, M.B., Ch.B., F.R.C.S.Ed., has commenced orthopaedic practice in partnership with Dr. A. C. Boonzaier at 312 Lister Buildings, Jeppe Street, Johannesburg. (*Telephones*: *Consulting Rooms*: 23-8410; *Residence*: Dial 655, ask for 1204).

* * *

Mr. L. Fatti and Mr. Paul Marchand have moved to the 6th Floor, Florence Nightingale Building, 22 Kotze Street, Hospital Hill, Johannesburg. (*Telephones*: 44-5388; 44-1955).

* * *

Prof. George Gee Jackson, M.D., Professor of Medicine at the University of Illinois, U.S.A., has arrived in South Africa on a short visit. He will lecture in Johannesburg, Durban and Pretoria to Medical School Faculties and students on such subjects as *Perspectives in Antibiotic Therapy, Ecology and Treatment of Antibiotic-Resistant Staphylococci* and *Diagnosis and Treatment of Pyelonephritis.*

* * *

Recent visitors to the Abbott Laboratories in Chicago were (*l. to r.*):

Dr. O. V. S. Kok, Professor of Anaesthesia, University of Pretoria and Dr. A. B. Bull, University of Cape Town. The two distinguished visitors were in the United States on a World Health Organization Travelling Fellowship in Anaesthetics. Shown here with them are Harold D. Arneson, President of the Abbott export companies and Thomas S. Bird, Vice-President.

## POST-GRADUATE REFRESHER COURSES

### MEDICAL GRADUATES ASSOCIATION—UNIVERSITY OF THE WITWATERSRAND

The next bi-annual refresher course will be held from 16 to 21 January 1961.

All intending applicants should apply to the Secretary of the Medical Graduates Association as soon as possible, at the Medical School, Hospital Street, Johannesburg.

* * *

## JOHNSON AND JOHNSON AWARDS FOR POST-GRADUATE CLINICAL STUDY IN SOUTH AFRICA

The following medical practitioners have each been granted one of these awards:

Dr. G. G. Airey, *Umtata;*
Dr. P. D. Beck, *Port Elizabeth;*
Dr. P. A. Goosen, *Vrede;*
Dr. J. A. Meyer, *Bethal;*
Dr. I. Sagor, *Worcester.*

* * *

Dr. Albert Rabinowitz, of Johannesburg, has been promoted to Associate Commander of the Order of St. John.

* * *

Dr. Ian Smith, B.D.S. (Rand), L.R.C.P., L.R.C.S. (Ire.), F.D.S. (Eng.), has commenced practice as a Maxillo-Facial Surgeon at 613 Harley Chambers, Jeppe Street, Johannesburg. (*Telephones*: *Rooms*: 22-0433; *Residence*: 43-4745).

* * *

Dr. J. H. S. Gear, Director of the South African Institute for Medical Research, has been invited as a World Health Organization Consultant, to advise on the initiation and organization of a diagnostic and research Virus Unit for East Africa.

* * *

## CHANGE OF ADDRESS

The following medical practitioners have moved to 516 van Riebeek Medical Buildings, Pretoria, and their telephone numbers remain unchanged, as follows:

Dr. H. J. Besselaar: 2-3007.
Dr. C. A. R. Schulenburg: 3-2841.
Dr. H. Aneck-Hahn: 2-5835.
Dr. Jan Lion-Cachet: 3-2040.

* * *

Prof. J. F. Murray, Deputy Director of the South African Institute for Medical Research and a member of the Executive Committee of the National Cancer Association of South Africa, recently left for overseas where he attended an Executive Committee meeting of the International Union Against Cancer. Prior to the U.I.C.C. meeting Professor Murray, under the aegis of the N.C.A.S.A., participated in a symposium on demographic aspects of cancer.

* * *

## NUTRITION SOCIETY OF SOUTHERN AFRICA

The 2nd Annual Congress will be held in the film auditorium, Shell House, corner of Smit and Hoek Streets, Johannesburg, from Monday to Wednesday, 7–9 November 1960.

The proceedings will include a symposium on *Minerals in Nutrition.*

Further information may be obtained from the Honorary Secretary (Dr. W. I. M. Holman), P.O. Box 395, Pretoria. (*Telephone*:—4-6011, Ext. 484 or 496).

Dr. H. H. Malherbe, of the Poliomyelitis Research Foundation and South African Institute for Medical Research, has left South Africa to visit various laboratories in the United States and Great Britain where he will discuss enteric virus research and the safety testing of poliomyelitis vaccine. Dr. Malherbe will return to South Africa about the middle of December.

* * *

Dr. H. E. van Hoepen, B.Sc., Arts (Leiden), M.D. (Clinical, Pretoria), has joined the partnership of Dr. Zalmon Wolf and Dr. M. C. Frame as a neurologist and psychiatrist, at Pasteur Chambers, Jeppe Street, Johannesburg. (*Telephones*:—*Rooms*: 23-8592; *Residence*: 46-9230).

* * *

The Division of Science Education of Syracuse University, with funds made available by the National Science Foundation, has invited Dr. C. G. Anderson, of the South African Institute for Medical Research, to spend the period from the middle of October to the end of December in the United States as a visiting lecturer at 10 universities. He will give talks to postgraduate students and senior science teachers on cell metabolism, bacteriophage, nucleic acid and protein synthesis, abnormal haemoglobins, and on immunity to bacterial infection.

Dr. Anderson will take the opportunity, on the return journey, of visiting various laboratories in Great Britain.

* * *

## VIII INTERNATIONAL CANCER CONGRESS

Every four years the International Cancer Congress is organized by the International Union Against Cancer. The next (eighth) meeting will be held in Moscow during the period 23–28 July 1962.

The following topics have been suggested for inclusion in the programme:

1. Role of viruses in the origin of cancer.
2. Precancerous lesions.
3. Tumour immunology.
4. Biochemistry of cancer.
5. Tumour-host relations.
6. The importance of achievements in experimental cancer research for clinical oncology.
7. New methods of cancer treatment (antibiotics, chemotherapy and hormonal therapy).
8. Geographical pathology of cancer.
9. The care of patients with advanced cancer.
10. Long-term results in the therapy of the most frequent types of cancer.
11. Cancer control.
12. Carcinoma of the stomach including the cardia.
13. Carcinoma of the lung.
14. Carcinoma of the ovaries.
15. Carcinoma *in situ* of the cervix.
16. *Varia* (the new most important developments in different fields of cancer research which were not included in the Programme).

The closing date for applications to submit reports and abstracts on papers is 1 December 1961.

All correspondence relating to the Congress should be addressed to:

Prof. L. Shabad,
General Secretary of the Eighth International Cancer Congress,
Academy of Medical Sciences of the U.S.S.R.,
14, Solyanka,
Moscow, U.S.S.R.

## DR. A. C. WATT

We deeply regret to record the accidental death of Dr. Andrew C. Watt, of Johannesburg.

* * *

## THE NUTRITION SOCIETY

### ADVANCE NOTICE OF 134TH MEETING

The following is the programme of a symposium to be held at the Middlesex Hospital Medical School, London, W.1 on Saturday, 17 December 1960 on

### CALORIE BALANCE IN MAN

Chairman: Sir Charles Dodds, F.R.S.
Dr. J. V. G. A. Durnin: *Physiological Principles.*
Dr. O. G. Edholm: *Environmental Factors.*
Dr. Raymond Greene: *Endocrine Factors.*
Prof. A. Kekwick: *Fat-mobility Factor.*
Dr. G. C. Kennedy: *Neurological and Psychological Factors.*
Dr. A. M. Thomson and Dr. F. Hytten: *Calorie Balance During Pregnancy and Lactation.*
Dr. E. M. Widdowson: *Energy Balance in Early Life.*

D. F. Hollingsworth,
(*Hon. Programmes Secretary*)

Ministry of Agriculture, Fisheries and Food,
Great Westminster House,
Horseferry Road,
London, S.W.1.

* * *

## THE MAURICE WEINBREN AWARD IN RADIOLOGY

1. This Award consists of a Certificate and a prize to the value of £25.
2. It will be made annually (in respect of a calendar year) for a published paper of sufficient merit dealing either with radiodiagnosis or radiotherapy.
3. The Award is restricted to medical practitioners registered in South Africa, but the paper may have appeared in any medical journal published in South Africa, or in the *British Journal of Radiology* or the *Journal of the Faculty of Radiologists*, London.
4. The Selection Committee may change or add to the names of the journals in which candidates may have published papers submitted for consideration.
5. Authors who wish to be considered for this Award must advise the Honorary Secretary of the Selection Committee to this effect by 31 December each year.
6. They must provide 6 copies of the paper submitted for consideration not later than the end of February in the succeeding year.
7. The Selection Committee consists of:
Prof. S. F. Oosthuizen;
Dr. Harris Jackson;
Dr. M. H. Fainsinger;
Dr. T. Fichardt;
Dr. J. N. Jacobson, and
Dr. H. A. Shapiro (*Acting Honorary Secretary*).
The address of the Acting Honorary Secretary is:
Dr. H. A. Shapiro,
P.O. Box 1010,
Johannesburg.
8. Members of the Selection Committee are not eligible for the Award.
9. The decision of the Selection Committee, in connexion with the making of an Award, is final and binding.

# MEDICAL PROCEEDINGS

# MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

P.O. Box 1010 · Johannesburg | Posbus 1010 · Johannesburg

*Editor : Redakteur*

H. A. Shapiro, B.A., Ph.D., M.B., Ch.B., F.R.S.S.Af.

Vol. 6 *Supplement* 19 November 1960 *Byvoegsel* No. 23


## ORAL POLIOMYELITIS VACCINE

### A Statement by the Secretary for Health

As is generally well known, the new type of living attenuated poliovirus vaccine is now available to local authorities from the Poliomyelitis Research Foundation. This vaccine has been approved for use by the Union Health Department after careful consideration and on the advice of its Virology Advisory Committee. This Committee recommended that the vaccine be used initially in organized and controlled vaccination campaigns by local authorities who are responsible for the health of the communities in their areas.

The vaccine is best used in the cool weather but, in order to prevent many cases of poliomyelitis which would otherwise occur during the ensuing summer, it has been decided that it would be wise to administer one dose of Type I vaccine at this time of the year as this is the type known to be responsible for extensive epidemics. The use of trivalent vaccine would have the effect of reducing the proportion of individuals in whom a satisfactory degree of immunity is reached as Type II tends to 'take' to the exclusion of Type I.

To achieve its purpose of preventing paralytic poliomyelitis in a community it is essential that the vaccine be given to as large a proportion of the susceptible population in as short a time as possible. A mass immunization campaign therefore has to be undertaken. To ensure the co-ordination of the campaign it is necessary that it should be under the direction and control of the local health authority responsible for the community. This is essential to ensure the best possible use in the shortest possible time of the somewhat limited supplies of the vaccine available.

It is hoped that private practitioners will support the efforts being made to immunize as many susceptible individuals as possible and in particular that they will advise parents in this connection.

All children and young adults, irrespective of whether they have received either a full or a partial course of immunization with the Salk type vaccine, or, even if they have had paralytic poliomyelitis, should be given oral vaccine. More specifically, all White South Africans between the ages of 6 months and 30 years, and immigrants up to the age of 40 from North-Western Europe and North America (who because of lack of exposure to previous immunizing infections are particularly susceptible) should be given oral vaccine. Non-Whites normally acquire a natural immunity at an early age and almost all adults and even older children are completely immune and do not need immunization. Therefore in these groups immunization is recommended up to school-leaving age.

Pregnancy at any stage is not a contra-indication to the use of the vaccine.

The vaccine should not be given to persons suffering from an acute illness, but it may be given to those with chronic infections, such as tuberculosis.

As practitioners may have some doubt about the administration of the vaccine to persons

known to be sensitive to penicillin and other antibiotics, it may be mentioned that the oral vaccine contains only minute amounts of these substances which, when taken by mouth, should not cause reactions.

Although it is intended to administer only one dose of Type I vaccine at this stage, to ensure the maximum degree of immunity of the population, it is planned to give all persons who have received this first dose 2 doses of trivalent vaccine during next winter. Normally, the intervals between doses of vaccine is 2 months or longer.

The vaccine is at present packed in ampoules containing 1,000 doses. It must be stored at a temperature of $-10°C$ to $-20°C$ and be kept at low temperatures until very shortly before use. Although smaller packings and even dilution for immediate use would be possible in the laboratory, the diluted vaccine must be used within 24 hours.

Although the vaccine is not being issued to private practitioners in the initial stages of the mass campaigns, it is the intention of the Union Health Department to consult fully with the medical profession, including the representatives of the private practitioners as well as the medical officers of health, to ensure the most effective and widest use of this vaccine during next winter and in subsequent years.

C.T. LTD